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STATISTICAL REVIEW OF FIVE-YEAR SURVIVALS AFTER SURGERY FOR CARCINOMA OF THE ESOPHAGUS AND CARDIAC PORTION OF THE STOMACH

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THE first systematic report on surgery of the esophagus and the cardiac portion of the stomach in Japan was presented by the late Dr. Seo and Dr. Ohsawa at the thirty-third Annual Congress of the Japanese Surgical Society in 1932. In those days radical surgery for carcinoma of the esophagus was one of the most difficult operations in this field and the operative mortality was high; Dr. Seo reported that, in 16 cases of carcinoma of the esophagus and the cardiac portion of the stomach exclusive of the cervical esophagus in which he performed esophagectomy, there were 8 operative deaths or an operative mortality of 50 per cent.

In the past 12 years in which I have been engaged in this field as a successor to Dr. Seo, the method of operation on the esophagus has been improved in many ways and fairly good results have been obtained in my department. I now have 35 cases of 5-year survivals of carcinoma of the esophagus and the cardiac portion of the stomach operated upon during this period. It would be significant to review the statistics and various operative methods from the standpoint of operative mortality and their far-reaching results.

During the past 12 years, from 1946 through 1957, 6,044 cases of carcinoma were observed in my outpatient clinic (Fig. 1).

Among male patients, carcinoma of the esophagus and the cardiac portion of the stomach and carcinoma of the stomach show an extremely high percentage. Each of them is approximately 45 per cent. Among female patients

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carcinoma of the stomach is also first, with a 43 per cent incidence. Carcinoma of the esophagus and the cardiac portion of the stomach follow, with an incidence of 26 per cent.

The total number of cases of carcinoma of the esophagus and the cardiac portion of the stomach was 2,382 (Fig. 2). Of these patients 1,591 were admitted to the hospital. Successful surgery was performed on 975 patients. The percentage of the operations on outpatients is 41 per cent, and that of the admitted patients is 61 per cent. However, this number includes 22 cases of carcinoma of the cervical portion of the esophagus, and these are excluded from the following reports.

Various operations were performed on these patients according to the location of the carcinoma (Table I).

TABLE I. OPERATIVE MORTALITY OF CANCER OF ESOPHAGUS AND CARDIA (Nakayama's Surgical Department, 1946-1957)

OPERATIVE METHODS			NO. OF CASES	OPERATIVE DEATHS	MORTALITY (%)
Carcinoma of upper and midthoracic esophagus	Antethoracic esophagogastrostomy		217	20	9.2
	R. intrathoracic esophagogastrostomy		38	6	15.8
	Esophagogastrostomy in neck (stomach in chest)		3	1	33.3
Carcinoma of lower esophagus and cardia	Intrathoracic	Esophagogastrostomy	136	7	5.1
		Esophagojejunostomy	56	2	3.5
	Abdominal	Esophagogastrostomy	95	3	3.2
		Esophagoenterostomy	368	8	2.1
		"Hanging up" method of esophgastro or jejunostomy	40	9	22.5
Total			953	56	5.8

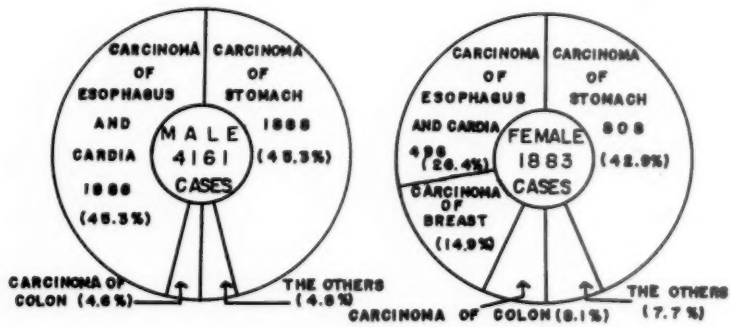

CARCINOMA OF ESOPHAGUS AND CARDIA 1888 (45.3%)
CARCINOMA OF STOMACH 1888 (45.3%)
MALE 4161 CASES
CARCINOMA OF COLON (4.6%)
THE OTHERS (4.8%)
CARCINOMA OF ESOPHAGUS AND CARDIA 498 (26.4%)
CARCINOMA OF STOMACH 808 (42.9%)
FEMALE 1883 CASES
CARCINOMA OF BREAST (14.9%)
CARCINOMA OF COLON (8.1%)
THE OTHERS (7.7%)

Fig. 1.—Statistics of 6,044 cancer patients seen as outpatients of Nakayama's Surgical Department, 1946-1947.

For carcinoma of the upper and middle portion of the esophagus, the following three different operations were used: (1) esophagectomy with antethoracic esophagogastrostomy; (2) esophagectomy with intrathoracic esophagogastrostomy; (3) esophagectomy with esophagogastrostomy in the neck with the stomach in the thoracic cavity (Fig. 3).

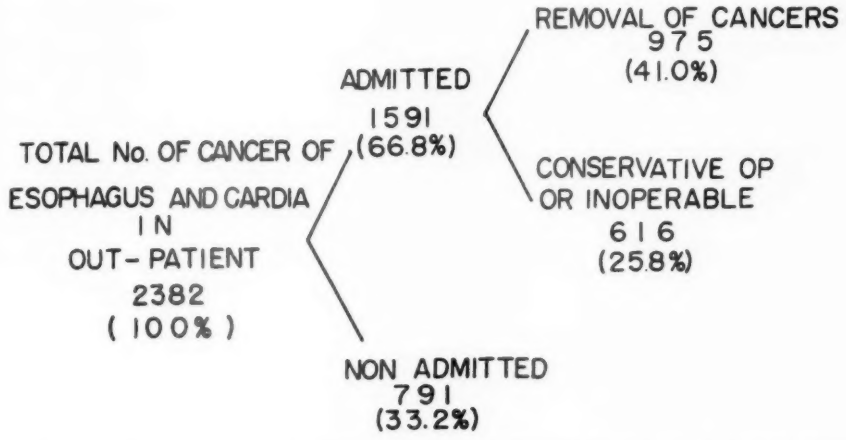
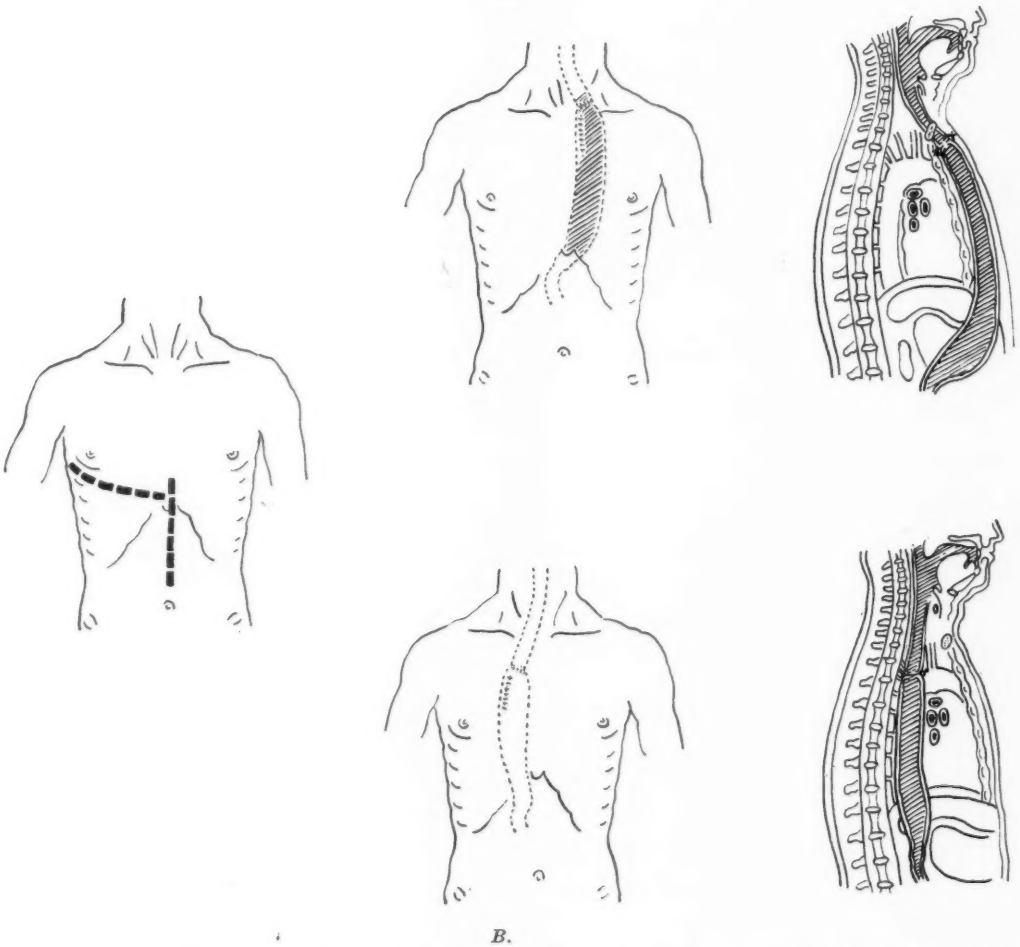


Fig. 2.—Statistics of carcinoma of esophagus and cardia, Nakayama's Surgical Department, 1946-1957.

A.



B.

Fig. 3.—Schematic illustration of both ante-thoracic (*A*) and right intrathoracic (*B*) esophagogastrostomy.

For carcinoma of the lower part of the esophagus and the cardiac portion of the stomach, the following five operations were used: (1) esophagectomy with left intrathoracic esophagogastrostomy; (2) esophagectomy and total gastrectomy with left intrathoracic esophagojejunostomy; (3) esophagocardiac resection with intra-abdominal esophagogastrostomy; (4) esophagectomy and total gastrectomy with intra-abdominal esophagoenterostomy; (5) esophagocardiac resection or total gastrectomy with abdominal incomplete anastomosis; adaptation of lower end of the esophagus and remaining part of the stomach, or jejunum, by hanging up and few sutures (so-called "incomplete anastomosis or hanging-up method").

Mortality of these operations for carcinoma of the upper and middle portion of the esophagus shows that antethoracic esophagogastrostomy is much better than intrathoracic anastomosis. For carcinoma of the lower part of the esophagus, intra-abdominal anastomosis shows better results than intrathoracic anastomosis. The "incomplete anastomosis or hanging-up method," which was used at the beginning of my study, is not in use now because of its high mortality.

Table II shows recent reports, each covering more than 100 cases, from all over the world, on carcinoma of the esophagus and the cardiac portion of the stomach. Surgical mortality in my department, 5.8 per cent, is much lower than that of others. The main reason for the lower surgical mortality is probably the difference in operation methods. In my department most cases of carcinoma of the upper and middle portion of the esophagus were operated upon with antethoracic anastomosis, while all others were done with intrathoracic anastomosis. Even in my department intrathoracic esophagogastroanastomosis shows higher surgical mortality, 15 per cent.

TABLE II. OPERATIVE MORTALITY OF CANCER OF ESOPHAGUS AND CARDIA
(Surgeons Reporting More Than 100 Cases)

SURGEONS	YEAR	NO. OF CASES	OPERATIVE DEATHS	MORTALITY (%)
Garlock	1954	180	57	31.7
Wu	1955	152	28	18.4
Sweet	1956	327	57	17.4
Alascio	1956	173	66	38.2
Nuboer	1956	195	—	—
Watson	1957	182	66	35.7
Petrov	1957	123	62	50.4
Lortat-Jacob	1957	308	102	33.1
Katsura	1957	177	25	14.1
Nakayama (including "hanging-up" method)	1958	953	56	5.8

Up to the end of 1957, 897 cases of carcinoma of the esophagus and the cardiac portion of the stomach were successfully operated upon in my department and 5-year survivals were 35 of 286 cases which were successfully operated upon from 1946 through 1952. The survival rate is 12.2 per cent. These are compared with recent reports reviewed (Table III).

TABLE III. POSTOPERATIVE SURVIVAL RATE OF CANCER OF ESOPHAGUS AND CARDIA (EXCEPT CANCER OF CERVICAL ESOPHAGUS)

SURGEONS	YEAR	CASES SUCCESSFULLY OPERATED UPON	2-YEAR SURVIVAL	5-YEAR SURVIVAL
Garlock	1954	123	27	11
Wu	1955	124	24	6
Macmanus	1956	64	—	5
Sweet	1956	270	83	24
Petrov	1957	61	—	6
Lortat-Jacob	1957	206	51	10
Other cases reported	1952-1957	—	—	21
Nakayama	1958	897	151	35
Total		—	—	118

Excluding carcinoma of the cardiac portion of the stomach, 87 cases of carcinoma of the esophagus were successfully operated upon from 1946 through 1952 and 5-year survivals were 16 cases, giving a survival rate of 18.4 per cent (Table IV). Ten of the 16 are patients with carcinoma of the upper or middle portion of the esophagus, and all of them were operated upon with antethoracic esophagogastrostomy, none, however, with right intrathoracic esophagogastrostomy. Survival rate curves after these two operations reveal the difference and the advantage of using antethoracic anastomosis (Fig. 4).

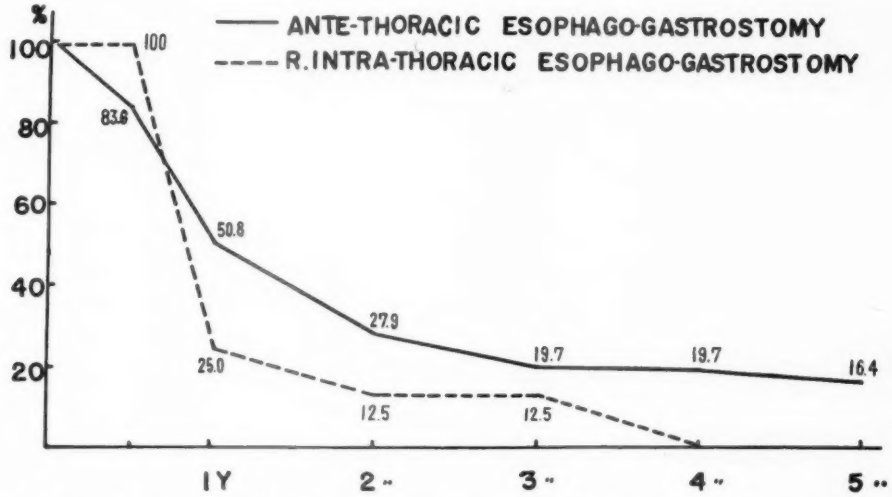


Fig. 4.—Comparison of survival rate between two types of esophagogastrostomy. Nakayama's Surgical Department (1958).

Comparing the 5-year survival rate with that of intrathoracic esophagogastrostomy by Sweet as reported in 1956, our 16.4 per cent is relatively better than the 6 per cent obtained by Sweet (Fig. 5). This may, again, chiefly depend on the difference in operation methods; that is, antethoracic anastomosis makes esophagectomy satisfactory and extirpation of lymph-node metastasis complete in carcinoma of the upper and middle portion of the esophagus.

TABLE IV. POSTOPERATIVE SURVIVAL RATE OF CANCER OF ESOPHAGUS
(Nakayama's Surgical Department, 1946-1952)

OPERATIVE METHODS	CASES SUCCESSFULLY OPERATED UPON	2-YEAR SURVIVAL	5-YEAR SURVIVAL
Antethoracic esophagogastrostomy	61	17 (27.9%)	10 (16.4%)
Intrathoracic esophagogastrostomy	20	5 (25.0%)	3 (15.0%)
Esophagogastrostomy in neck (stomach in chest) and "hanging-up method"	6	4 (66.7%)	3 (50.0%)
Total	87	26 (29.9%)	16 (18.4%)

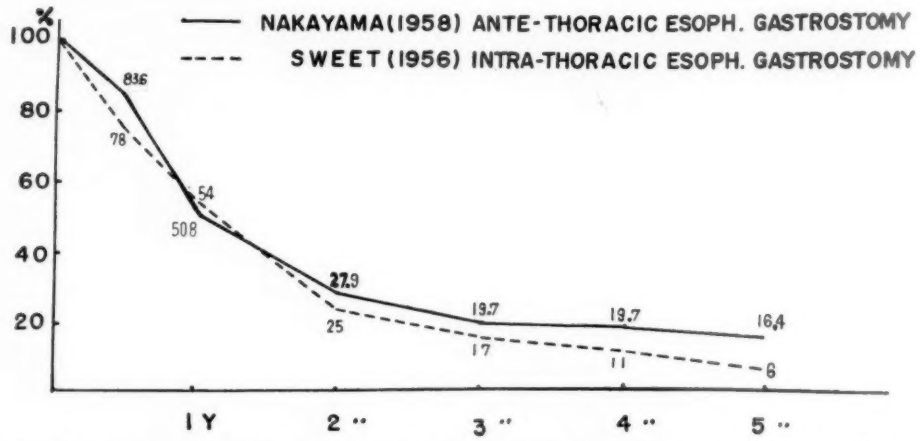


Fig. 5.—Comparison of survival rate between two types of esophagogastrostomy: Nakayama's Surgical Department (1958) and Sweet (1956).

SUMMARY

1. Statistical study is undertaken on carcinoma of the esophagus and cardiac portion of the stomach as seen in Nakayama's Surgical Department from 1946 through 1957.

2. During this period, successful surgery was performed in 953 cases of carcinoma of the esophagus and cardiac portion of the stomach exclusive of carcinoma of the cervical esophagus, and operative deaths were 56, making an operative mortality of 5.8 per cent.

3. Five-year survivals after successful operation of carcinoma of the esophagus and cardiac portion of the stomach from 1946 through 1952 were 35 of 286 cases and the survival rate was 12.2 per cent. These cases includes 16 cases of carcinoma of the esophagus exclusive of carcinoma of the cervical esophagus and cardiac portion of the stomach. Ten cases were carcinoma of the upper or middle portion of the esophagus and the survival rate was 16.4 per cent.

4. From the point of operative mortality and long-term results, antethoracic esophagogastroanastomosis showed better results than intrathoracic anastomosis.

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RETROSTERNAL ARTIFICIAL ESOPHAGUS FROM JEJUNUM AND COLON

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IN 1907 P. A. Herzen[9] was the first surgeon in the world to complete an operation for benign stricture of the esophagus on a 20-year-old girl.

Herzen performed his operation after the method of Roux (Lausanne),[23] who 9 months earlier (June 12, 1906) had done the initial stage of the operation on an 11-year-old boy. Roux, however, delayed attempts at the second stage of the operation—the anastomosis in the neck—another 4 years (1911).

Some years later, after the successful outcome of Herzen's operation, surgeons often had fatalities following the operation.

Further research was aimed at reducing the risk of necrosis of the jejunal loop. At the same time it was suggested to extend the jejunal loop subcutaneously as high as possible in the neck.

S. S. Yudin (Sklifosovsky Institute, Moscow)[28-30] did more work than anyone else in this field. For 20 years after 1928, he improved the Roux-Herzen operation considerably and performed it on more than 300 patients.

Following Yudin's work, we at the Sklifosovsky Institute with G. R. Khundadze devised a new method. It consists of the mobilization of the mesenteric root of the jejunum. If the transposition of the jejunal loop to the chest cannot be raised higher than the clavicle, then the cecum must be raised and the parietal peritoneum under it is dissected. This posterior leaf of the peritoneum is also dissected medially toward the spine. Thus, the root of the mesentery is freed and it is then possible to raise it upward with the whole mass of associated jejunal loops. Without risk to the vitality of the jejunum loop, it is possible by this simple method to transfer it 10 to 20 cm. higher than by ligature of the mesenteric vessels alone.

We have performed this operation more than 150 times with unfailing success since 1949.

Seven months after publication of our work,[19] Dubost and Bernier[5] also described the operation, using a similar technique.

However, the transference of the jejunal loop subcutaneously is not satisfactory to many surgeons. Methods of intrathoracic operation have been introduced. At first the intrapleural method was used. The mobilized jejunal loop was transferred through a hole in the diaphragm and anastomosed with the esophagus inside the intrapleural cavity. This method has a limited application since it is used when there is a stricture of the lower part of the esophagus. There is no need to separate a long jejunal loop, which simplifies the operation.

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The description of the retrosternal method of operation (through the anterior mediastinum) was published by Robertson and Sarjeant[21] and by N. I. Eremeev[6] 6 months later. It is applicable in most cases of extensive cicatrization of the esophagus, when the stricture is found not only in the thoracic part of the esophagus, but also in the neck. In such cases it is necessary to transfer a long intestinal loop.

The retrosternal esophagus meets the aesthetic requirements more than the subcutaneous one, especially with young patients. Besides, the jejunal loop, which had been transferred behind the sternum, functions better than the subcutaneous one.

For the last 5 years this method has been popular in Europe and the United States.

To date, we have performed 78 retrosternal esophagoplasties on patients with benign strictures as a result of swallowing caustic solutions.

A jejunal loop was used in 55 patients and the right or the left half of the colon was used in 23 instances.

TABLE I

METHOD OF OPERATION	YEARS	NUMBER OF CASES		PER CENT
		TOTAL	DIED	
Antethoracic (jejunal) (S. S. Yudin)	1942-1948	256	24	9
Antethoracic (jejunal) (B. A. Petrov, D. A. Arapov, B. S. Rosanov, G. R. Khundadze and others)	1949-1953	252	11	4.3
Retrosternal (jejunal) (B. A. Petrov)	1952-1958	55	4	7.2
Retrosternal (colic) (B. A. Petrov)	1956-1958	23	2	9.5

RETROSTERNAL ESOPHAGUS FROM JEJUNUM

In the retrosternal plastic operation the jejunal loop is introduced through the anterior mediastinum directly in the midline, dividing the friable cellular tissues between the pleural cavities. The dissection of the pleura from either side should be carefully done in order not to tear the pleura; otherwise the loop will be drawn into the pleural cavity under the influence of negative pressure and it will kink. This would not be favorable for functioning of the jejunal loop and would be dangerous for its viability. If there are doubts concerning the occurrence of pneumothorax, a roentgenogram should be made after the operation and, if necessary, air should be aspirated.

Only one of our patients, a girl of 7, died as a result of bilateral pneumothorax. Her death was owing to failure to recognize the nature of the postoperative complication.

Another more dangerous complication is necrosis of a part or the whole of the jejunal loop in the retrosternal cavity. It is far more dangerous than necrosis of a subcutaneous jejunal loop. It is easier to recognize it subcutaneously, and it can be removed without difficulty. In the retrosternal

space the surgeon cannot see the jejunal loop or palpate it. It is easy to miss the development of necrosis and thus be late in removing the jejunal loop. In 1953 we lost 3 patients due to necrosis of a part of the jejunal loop.

The indications of necrosis of the tiniest bit of the jejunal loop in the retrosternal cavity are only indirect: such as high temperature and increasing pulse rate. Roentgenography is of primary importance. If during the first days after the operation the jejunal loop behind the sternum is distended and contains fluid levels, it is obvious that ileus of the loop has occurred. Then it is necessary to open the neck wound and examine the upper part of the jejunal loop. If necrosis is observed, it is necessary to remove the jejunal loop from the tunnel and to transfer it subcutaneously. We have done this in 7 cases. Later, good subcutaneous esophagi were made for these patients. In 3 patients, after necrosis of the edge of the jejunal loop at the neck, the intestinal stroma had closed and, in order to open it, we had to excise a part of the manubrium sterni. In these cases functioning results were good.

Fig. 1.—The blood supply of mesentery in the initial part of jejunum.

In order to obviate similar complications, transfer of the jejunal loop through the anterior mediastinum is used only in cases with perfectly good blood supply to the loop. In doubtful cases, when the jejunal loop has turned blue after the ligation of the vessels, it is safer to transfer it subcutaneously. Later, when it is obvious that the loop is viable, it is dissected from the subcutaneous tunnel and transposed retrosternally. We have done this in 8 cases. However, in the last 22 retrosternal operations, we have had no complications.

The operation is performed in three stages. First a long jejunal loop is brought out by the Roux-Herzen-Yudin method. If it is impossible to draw it up to the neck, our method of mobilization of the mesenteric root is applied.

The loop is transferred retrosternally to the neck and its end is left subcutaneously. The second stage, anastomosis of the jejunal loop with the esophagus, preferably side-to-side, is performed after 3 to 5 days. In case the stricture is situated very high, it is necessary to perform a lateral pharyngostomy and to connect it with the jejunal loop with the aid of skin flaps. We have had 10 patients on whom we had to perform pharyngostomy.

These operations took much time to complete (3 to 4 months), but all patients recovered with good functional results. The third stage, anastomosis of the jejunal loop with the stomach, is performed in 3 to 6 months after discharge from the clinic, when good functioning results of the artificial esophagus are noted. Laparotomy is then repeated and the jejunal loop is freed from adhesions, divided, and anastomosed end-to-side with the stomach. The distal end of the jejunal loop is closed with a purse-string suture. It is often necessary to anastomose this end with the extracted jejunal loop, for which purpose the former Y-like anastomosis is divided. Thus, the primary anatomic correlations are reestablished. It is necessary to include the stomach in the act of digestion; otherwise the patient will suffer nutritional disturbances. Sole jejunal nutrition sometimes causes protein loss.

Our experience shows that retrosternal esophagus from jejunal loop has great advantages. It functions perfectly enough that the patient's diet is unrestricted and he is able to return to work. During the last few years in our clinic this method has been used more frequently than subcutaneous and intrapleural placement of the neoesophagus.

RETROSTERNAL ESOPHAGUS FROM COLON

Although the artificial esophagus made from a jejunal loop is good, there is always some danger the extracted loop will not retain its viability. The history of the development of these operations confirms this.

That is why the operation proposed in 1911 by Kelling[10] and Vulliet[26] consisting of a transposition of a part of the colon is again being applied in recent years.

In 1934 Ochsner and Owens[16] gathered descriptions of 20 cases of subcutaneous reconstruction of esophagus made of colon from the literature. Beginning in 1951 a new series of communications[12, 13, 25] has appeared. Recently more than 50 operations of artificial esophagus made from the left or right half of the colon, including retrosternal ones, were described in the literature.

For the most part the colon has an excellent blood supply, which is not at all disturbed if only the ileocolic artery and vein are ligated for the right half and the left colic artery for the left half of the colon.

We transferred the colon retrosternally in 23 patients with esophageal stricture and lost two.

All operations were performed after an accidental swallowing of caustic solutions. The patients arrived from distant regions of our country, with gastrostomy and total obstruction of the esophagus.

TRANSPOSITION OF THE RIGHT COLON

In mobilizing the cecum and ligating only one trunk of the ileocolic artery, it is possible to mobilize the cecum, the ascending colon, and a part of the ileum 20 cm. in length at the mesenteric border. The blood supply is maintained by the large trunk of the mid-colic artery. Sometimes it is so good that one can see pulsations at the end of the colon, even after its transfer to the neck. This is never observed with the jejunal loop. Appendectomy is obligatory. The re-establishment of the continuity of the intestinal tract after the transposition of the colonic loop is restored by aid of a lateral ileotransverse anastomosis.

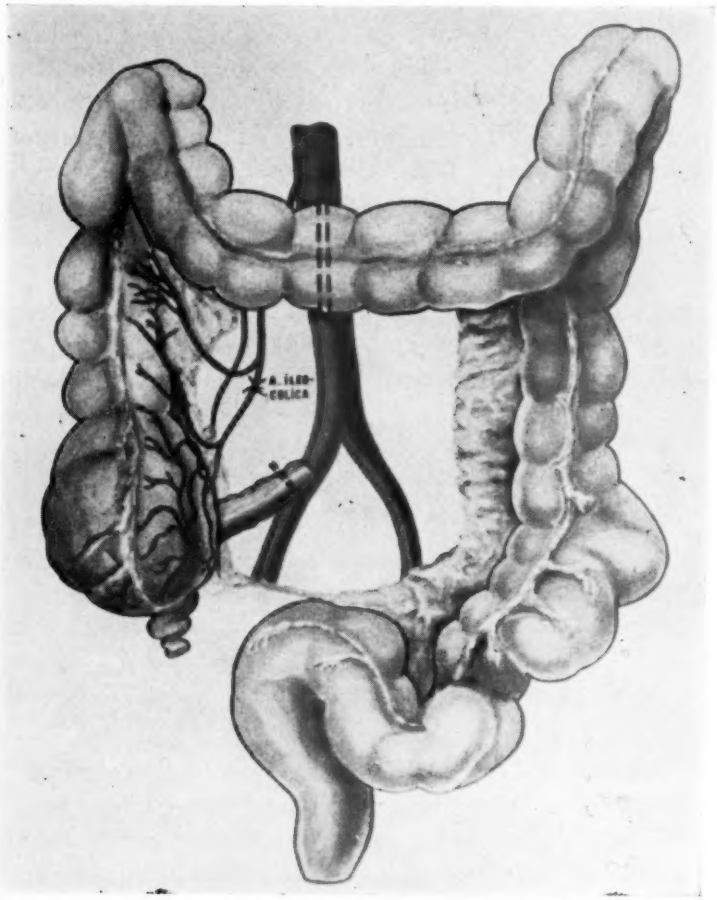

Fig. 2.—Transposition of the right half of the colon. The lines of dissection of the jejunal loop and transversal are shown.

The right half of the colon is transferred behind the sternum isoperistaltically so that the cecum is left retrosternally and the part of the ileal loop is brought out in the neck. The transposition of the right half of the colon behind the sternum was performed on 15 patients with no deaths.

TRANSPOSITION OF THE LEFT COLON

If it appears by laparotomy that the left half of the colon is of sufficient length, it may be used for making the artificial esophagus. For this purpose

the splenic flexure of the colon and the descending colon are mobilized and only the left colic artery is ligated. As in the right half of the colon, the large trunk of the mid-colic artery is preserved to insure a satisfactory blood supply.

After intersecting the transverse colon and the initial part of the sigmoid flexure, the mobilized part is transferred upward in an antiperistaltic direction. The continuity of the intestinal tract is restored by end-to-end anastomosis.

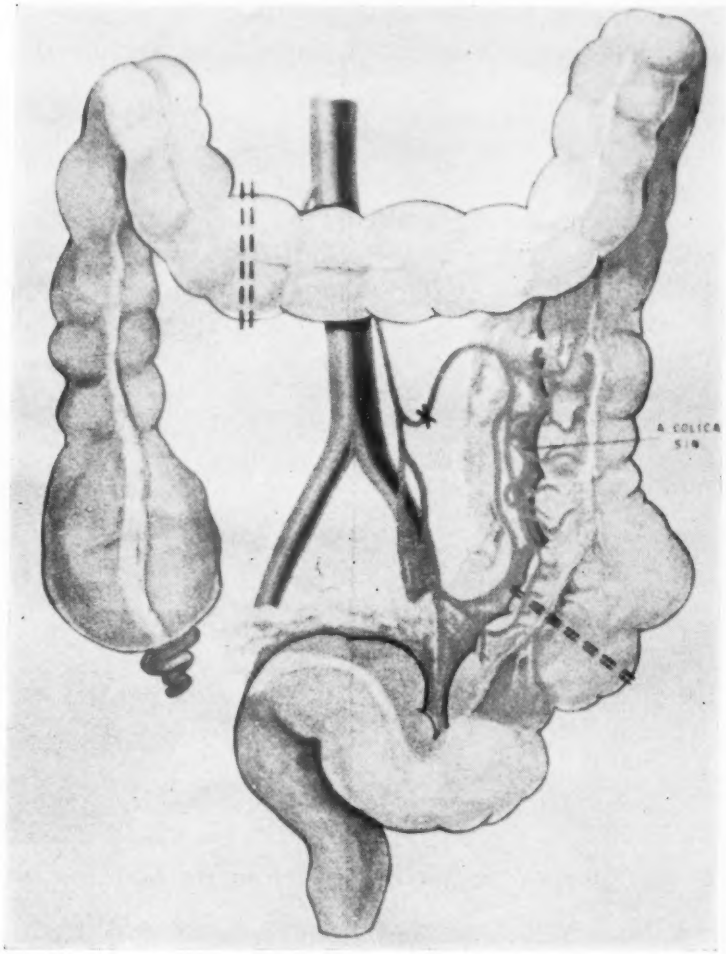

Fig. 3.—Transposition of the left half of the colon. The lines of dissection of the left half and transversal are shown.

We have completed 8 operations with the left half of the colon transferred into the anterior mediastinum with two fatalities. One male patient died from rupture of the end-to-end anastomosis, although neomycin was used; one young girl died from subdiaphragmatic abscess after rupture of the gastrocolic anastomosis. Evidently, the pathogenic microflora is of great importance. In creating an artificial esophagus from a jejunal loop, we never had similar complications in several hundred cases. Unfortunately, the left half of the colon cannot be used on all occasions. Four times we could not bring it out

to the neck. It was too short and the distribution of the vessels was not convenient. The mortality rate analysis shows also that end-to-end anastomosis of the colon is more risky than ileotransverse or than the jejunal anastomosis.

There are several methods of transposition of the colon. The operation can be completed in one stage or it may be divided into several. We have tried different methods and prefer to perform the operation in several stages.

The simplest method consists of transfer of the right half of the colon with the cecum and a part of the ileal loop into the anterior mediastinum followed by the ileotransverse anastomosis, without intersecting the transverse colon.

At the second stage the plastic operation is performed in the neck, which is sometimes difficult to do when there is a stricture of the proximal esophagus. When normal swallowing is established through the mouth, we proceed to the third stage: the laparotomy is repeated, and the transverse colon is divided and anastomosed with the stomach. The distal end of the transverse colon is closely oversewed because of the ileotransverse anastomosis. It stands to reason that in dividing the transverse colon it is necessary to avoid ligating the vessels of the mesentery.

Fig. 4.—The left half of the colon is brought out and put on the chest.

When using the left half of the colon, the operation is also divided into stages. The colon is drawn up to the neck in an antiperistaltic direction. Therefore it is necessary to divide the loop immediately and to anastomose it with the stomach. The gastrostomy must be closed, which is a disadvantage, since a fistula might open in the neck. Therefore at the first stage also the lower end of the colon could be tightly closed. Afterward, when the plastic operation is completed in the neck, the laparotomy is repeated and the colon is anastomosed with the stomach.

What are the functional results of the artificial esophagus made from jejunum and colon?

The retrosternal esophagus from jejunal loop after the operations were completed shows perfect functioning results and it seems nothing else could be

desired. But sometimes there are too many loops and roentgenography shows that they collect in the lower part of the tunnel, which slows down the passage of food.

The creation of esophagus from the right half of the colon with a part of jejunal loop is unusually attractive due to its technical possibilities. The operation proceeds perfectly. The blood supply remains intact. The ileotransverse anastomosis offers little risk of rupture. But it appears that the ileocecal valve does not help but even hampers the process. Evidently, it cannot accommodate the passage of a compact lump of food, since even in normal conditions only liquid passes through.

Indeed, some of our patients complained of spasms and stopping of food in the region of the ileocecal valve. Although the left half of the colon is transferred in an antiperistaltic direction, it is thinner than the right half, is straighter, and is easily brought out to the neck between the ends of the sternocleidomastoid muscles.

Clinically and roentgenologically the left colon functions perfectly, even better than the jejunal loop. It is true that we have had only 5 patients under observation using the left colon to date, but all have good functional results. Unfortunately, when transferring the right as well as the left half of the colon, fistulas in the neck may appear, which heal more slowly, at least not as well as with a jejunal loop. However, not one patient has complained of a bad taste in the mouth or belching.

So, we arrive at the conclusion that, if the blood vessels of the mesentery of the jejunum are favorably placed and when the risk of necrosis is lowered as much as possible, it is more favorable to use a jejunal loop for the retrosternal operation from the functional point of view.

The left half of the colon is preferable to the right one. It functions well. Unfortunately, owing to its shortness, its mobilization is not always possible.

The mobilization of the right half of the colon with a part of jejunal loop is nearly without risk of necrosis because of its perfectly good blood supply even at its highest transposition to the neck. However, the functional results of jejunal loop are better. This is due to the bad influence of the ileocecal valve. It is necessary to find some means to do away with this obstruction.

The possibilities of creating an artificial esophagus have certainly increased. We are continually nearing the possibility of making the retrosternal operation less risky and achieving more perfect functional results.

SUMMARY

An analysis is given of 78 operations of retrosternal artificial esophagus, created from a jejunal loop in 55 patients and from the colon in 23 patients. Functional results are reviewed.

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USE OF INTACT OMENTUM FOR CLOSURE OF FULL-THICKNESS ESOPHAGEAL DEFECTS

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THE greater hazard of operative procedures carried out on the esophagus in comparison with similar efforts involving the stomach and intestine is well known. This greater risk is due largely to an increased incidence of anastomotic suture line leakage and disruption. The nature of the predominantly longitudinal esophageal musculature and the absence of a serosal coat contribute to this diminished suture line integrity. The more tenuous esophageal blood supply and the absence of potentially reinforcing structures, such as omentum and adjacent intestine, are also factors.

The usefulness of omentum in reinforcing questionable intestinal suture lines and in walling off and localizing potentially dangerous inflammatory processes and gastrointestinal perforations has been recognized by surgeons for many years. A recent study from this laboratory by Hardin, Su, and Battersby[2] has demonstrated the suitability of a plug of intact omentum for the closure of an open duodenal stump in dogs following partial gastrectomy and gastrojejunostomy.

Thompson and Pollock[5] and Westermeyer[7] have reported the use of free omental grafts in esophageal surgery. Holman and McSwain,[3] in 1942, described the clinical and experimental utilization of intact omentum to reinforce anastomoses between the stomach and esophagus. The present study was carried out to evaluate the suitability of intact omentum for the closure of relatively large full-thickness defects of the lower esophagus. The omentum was brought into the chest through an opening in the diaphragm.

MATERIAL AND METHODS

Healthy mongrel dogs were used. An effort was made to limit the oral intake of liquids and solid food for a short period prior to operation. Intravenous Pentothal was utilized for anesthesia. Oxygen was administered through an endotracheal tube by means of a mechanical respirator. The lower left pleural cavity was entered through an intercostal incision. The lower esophagus was mobilized and secured between Potts coarctation clamps. An oval full-thickness defect of standard size, measuring approximately 2.5 by 2 cm., was made by resecting a segment of esophageal wall (Fig. 1). The

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Potts coarctation clamps were useful not only for manipulation of the esophagus, but also in the control of bleeding along the margins of the defect and the prevention of possible contamination of the pleural cavity and area of operation by gastric and upper esophageal contents.

The tendinous portion of the diaphragm was opened and a segment of intact, viable omentum was brought into the left pleural cavity. The segment of omentum was secured to the margins of the defect and the adjacent esophageal musculature by interrupted sutures of 5-0 arterial silk. No effort

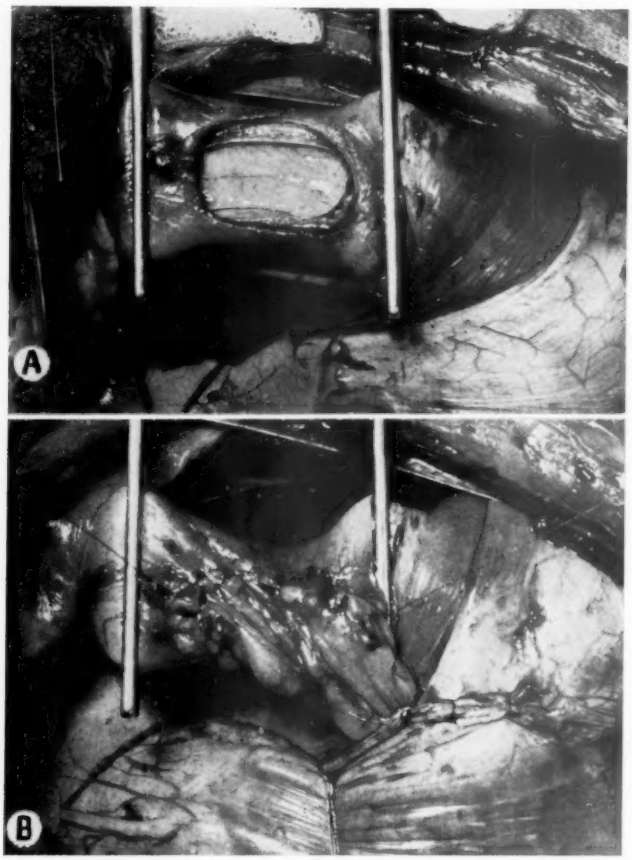


Fig. 1.—Photographs showing the standard type of esophageal defect created (*A*) before, and (*B*) after closure with intact omentum brought through a hole in the diaphragm. The diaphragm is closed around the omental pedicle with interrupted silk sutures.

was made to thrust omentum into the lumen of the esophagus. Upon completion of the closure and release of the occluding Potts coarctation clamps, the opening in the diaphragm was narrowed with interrupted sutures of 3-0 silk to an area large enough to permit only the passage of the omental pedicle without undue constriction. The omental pedicle was secured to the margins of the newly created diaphragmatic hiatus by several interrupted sutures of 5-0 arterial silk. The left thoracotomy incision was closed in layers and a catheter left in place during the closure was aspirated and withdrawn.

The animals were given nothing by mouth for the first 24 hours after operation. On occasion, intravenous fluids were given during this period. For the following 10 days, they were given only milk by mouth. A soft meat diet then was given for one month prior to the resumption of a regular diet. Intramuscular penicillin, 200,000 units a day, was given for the first 5 days after operation.

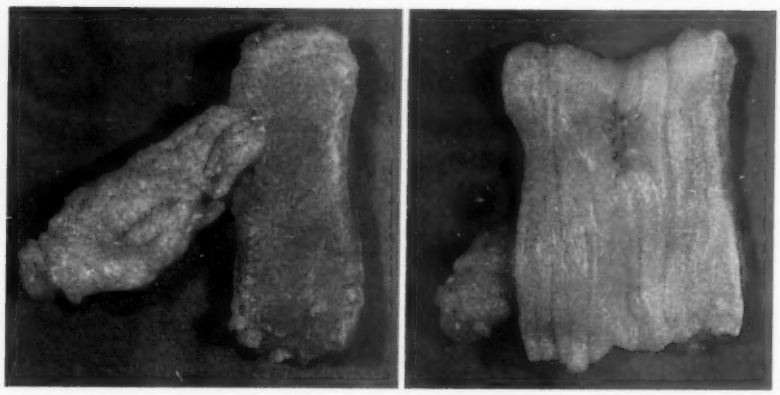
RESULTS

The operation was carried out, as described, in 39 consecutive animals. Conditions were encountered at the time of operation in 7 animals which led to the designation of these animals as poor risks and the procedures as unsuitable tests of the adequacy of omentum for the closure of the type of esophageal defects created in this study. The difficulties in these animals were due either to inadequate preoperative preparation with excessive amounts of solid food or liquids in the stomach and esophagus, or to an abnormally small amount of available omentum to close the defects without undue tension. These animals were designated poor risks at the time of operation and the results in this group will be considered separately.

The operation was regarded as a satisfactory test of the omental closure in 32 dogs. The closure was successful with survival of the animal in 28. One animal died in the early hours after operation due to inadequate re-expansion of the lungs with pneumothorax. Three animals died with empyema from 2 to 7 days following operation and were regarded as failures. However, gross disruption of the omental closure was not discovered in any of the 3.

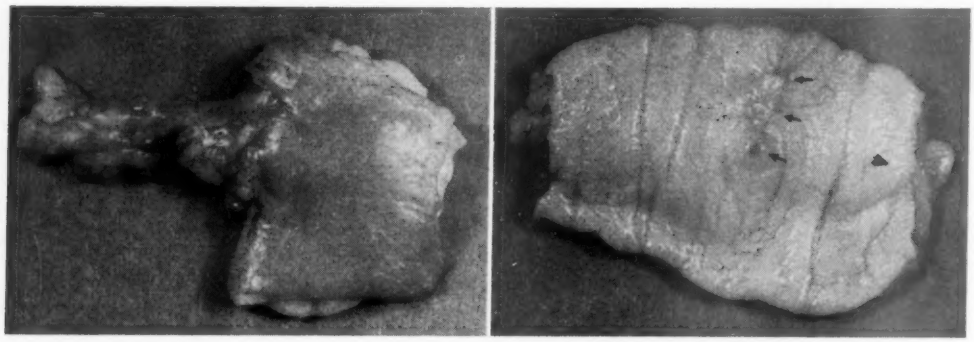
The appearance of the defect closure and the nature of the healing process were investigated by postmortem examination at varying intervals following operation in those animals with successful defect closures. Except for 1 animal in which a sponge had inadvertently been left behind at the time of defect closure, empyema was not encountered in this group. The defect closure was examined from 10 to 21 days after operation in 5 dogs, from 28 to 45 days in 9, from 52 to 91 days in 7, and from 96 to 112 days in 7. A progressive narrowing and contraction of the defect with the passage of time were noted (Figs. 2, 3, and 4). The omental plug sealed the defect satisfactorily and a significant diminution in the size of the defect was produced in the early days and weeks after operation. Healing generally appeared to be complete, grossly, between 2 and 3 months following defect closure. At times, some residual defect remained as a small rectangular area of omental plug covered with epithelium. More often, only an irregular linear or stellate scar remained. Reduction or distortion in the size of the esophageal lumen at the site of defect closure was not found.

Of the 7 poor-risk animals, designated at the time of operation as unsuitable tests of the procedure, 4 died from 2 to 14 days after operation with empyema. Despite the unfavorable circumstances, the defect closure was successful with survival of the animal in 3. One dog was alive and doing well



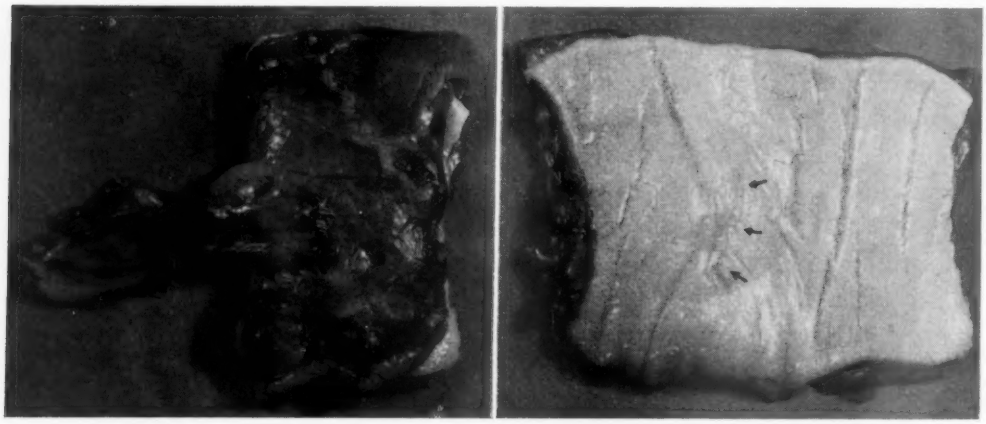
A. *B.*

Fig. 2.—Photographs before (*A*) and after (*B*) opening of the autopsy specimen of esophagus 1 month after operation. The omentum has become firmly attached to the esophagus and considerable contracture of the closed defect has occurred. A few sutures are visible in the area of defect closure.



A. *B.*

Fig. 3.—Appearance of esophageal defect closure with intact omentum at autopsy 2 months after operation with the esophagus closed (*A*) and open (*B*). The area of defect closure (*arrows*) has become contracted and epithelized.



A. *B.*

Fig. 4.—Photograph showing postmortem appearance of esophageal defect closure with esophagus closed (*A*) and open (*B*) 3 months following operation. The defect closure area may be seen from the lumen as an irregular, stellate scar (*arrows*).

when examined 3 weeks following operation. This animal subsequently disappeared from the colony. Adequate defect closure was observed at postmortem study in the other 2 animals, 42 and 56 days after operation. A small granuloma was found in the area of defect closure in one of them.

Disruption of the closure of the diaphragm with the formation of a rather significant diaphragmatic hernia was encountered at autopsy in 6 animals. This occurrence was most likely due to undue tension from the omentum on the greater curvature of the stomach, coupled with inadequate closure of the diaphragm. Five of these 6 diaphragmatic hernias occurred in animals operated upon during our early experience with the procedure. Greater care in closure of the diaphragm has eliminated this complication in subsequent animals.

DISCUSSION

Perhaps the greatest use of intact omentum has been in attempts at "revascularization" of ischemic organs and for closure of defects and perforations in the gastrointestinal tract. It has been employed in efforts to achieve renal "revascularization" in the management of hypertension presumed to be due to renal ischemia and to obtain hepatic vascular communications in the control of portal hypertension due to hepatic cirrhosis. Walters,[6] in 1937, reported the successful use of a flap of intact omentum in the closure of rectovaginal fistulas in 2 cases. The successful extra-abdominal use of a rather large segment of intact omentum on a pedicle to cover a large, traumatic forearm defect was described by Cannaday.[1]

The intrathoracic use of intact omentum has been recorded by O'Shaughnessy[4] and others in attempts to bring about myocardial revascularization in the management of coronary arterial insufficiency. While the adequacy of the myocardial revascularization resulting from cardio-omentopexy may be questioned, this work did demonstrate that a rather sizable amount of intact, viable omentum could be brought into the chest through a small opening in the diaphragm for use in that area.

The use of free omental grafts in esophageal surgery has already been mentioned. Thompson and Pollock applied free omental grafts experimentally in dogs to a variety of intrathoracic organs including pleura, lung, bronchus, esophagus, vena cava, and aorta and reported a high incidence of "takes." Free omental grafts were used by them to reinforce end-to-end anastomoses of the lower esophagus in 5 dogs. Suture line leakage occurred in one of the 5.

Holman and McSwain described the use of intact omentum in one case to reinforce an esophagogastric anastomosis. This experience prompted them to investigate the suitability of intact omentum in the experimental reinforcement of esophagogastric anastomoses carried out with the Murphy button. Reinforcing omentum was not used in a group of 9 dogs and a leak occurred in 5 of them, whereas a leak was encountered in only one of 7 dogs in which reinforcing omentum was employed.

The experience in this study suggests that intact, viable omentum, brought on a pedicle through the diaphragm, may be used for the closure of relatively

large full-thickness defects of the lower esophagus with expectations of success. It is of interest that the omentum was secured to the margins of the defect with a limited number of interrupted sutures and that no effort was made with the placement of the sutures to secure a water-tight closure of the defect. The omentum was not thrust into the lumen of the esophagus but was secured to the margins of the defect and the adjacent esophageal musculature. Successful closure of the defect without empyema was achieved in 28 of 31 definitive, adequate test operations. Success also was obtained, somewhat surprisingly, in 3 of 7 dogs that were designated at the time of operation as poor risks and inadequate tests of the procedure, due to grossly unsatisfactory preoperative preparation of the animal or insufficient amount of omentum for defect closure without undue tension. Even in the small number of animals that died of empyema and were regarded as failures, no disruption of the omental closure could be demonstrated. These findings would appear to suggest that the use of intact omentum for the closure of esophageal defects and the support of esophageal anastomoses and closures of dubious integrity is worthy of clinical trial.

SUMMARY

The literature pertaining to some of the uses of intact omentum is reviewed briefly. The experimental use of intact omentum to close relatively large full-thickness defects in the lower esophagus in dogs is described. The omentum was brought into the left chest through a hole in the diaphragm and was secured to the margins of the defect and to adjacent esophageal musculature with interrupted sutures of fine silk. This method of esophageal defect closure proved to be surprisingly effective. It was employed in 39 consecutive dogs. Successful defect closure with progressive healing and absence of empyema was achieved in 28 of 31 suitably prepared dogs with an adequate amount of omentum. The nature of the failures and the experiences with the poor-test-of-operation group are discussed. Defect healing occurred with progressive diminution in defect size. From these observations, the clinical use of intact omentum for the closure of esophageal defects and the support of questionable esophageal anastomoses would appear to be justified.

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EVALUATION OF RESPIRATORY FUNCTION IN SURGICAL PATIENTS: IMPORTANCE IN PREOPERATIVE PREPARATION AND IN THE PREDICTION OF PULMONARY COMPLICATIONS

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ATTENTION to well-known details of postoperative care has resulted in a decreased incidence of pulmonary complications.[1] Such complications, nevertheless, continue to be a significant cause of postoperative morbidity and mortality in all major surgery. A reasonable way to reduce them further lies in the preoperative detection and correction of ventilatory insufficiency. Although history, physical examination, and x-ray examination of the chest may suggest such insufficiency, evaluation based on these measures alone may fail to detect significant ventilatory defects in some patients.[4, 8] Two recently proposed methods which do provide a simple and accurate evaluation of pulmonary function are the cough analysis developed by Greene[3] and the ventilatory function testing system of Miller.[6] These methods correlate well with "pulmonary risk." This term will be used throughout this paper to refer to a patient's chance of developing postoperative pulmonary complications.

The purpose of this report is to outline a simple program of preoperative pulmonary evaluation and therapy which employs these two methods. Case reports illustrating the value of this program will be presented.

THE PROGRAM

Regimen for Preoperative Pulmonary Evaluation.—When first seen, all surgical patients are questioned specifically regarding smoking habits and for a history of exertional dyspnea, asthma, chronic or morning cough, and respiratory infection. At the time of initial physical examination note is made of the anteroposterior diameter of the chest and the velocity and volume of chest excursion. Posteroanterior and lateral x-rays of the chest are obtained.

All patients are then subjected to cough analysis by the method of Greene.[3] This is performed by observing a voluntarily induced cough which normally is single, brief, and dry. Induced coughs may be abnormal in one or both of two ways: they may be self-propagating or they may be wet. The former refers to the involuntary paroxysms which follow the initial voluntary cough; the latter refers to the productivity of the cough. An abnormal cough is rated on a scale of one to four according to these characteristics.

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Patients with a history suspicious of pulmonary disease, patients with abnormal physical examinations or x-rays, or patients with abnormal induced coughs receive the quantitative pulmonary evaluation of Miller.[6] This ventilatory function testing system is based on two simple determinations: vital capacity (V.C.) and 0.5 second expiratory capacity (0.5 sec. E.C.). Both of these are determined by having the patient expire forcibly from a position of maximum inspiration and measuring the amount expired in the first 0.5 second (0.5 sec. E.C.) and the total amount expired (V.C.). Both values can be obtained with a Collins respirometer* or a Gaensler-Collins timed vitalometer.*[2] These determinations require a minimum of patient cooperation and can be conducted by a technician. To relate the patient's function to predicted norms, these two values are plotted as the ratio of the patient's vital capacity to the predicted vital capacity and as the ratio of the patient's 0.5 second expiratory capacity to the measured vital capacity. These two parameters have been chosen because all ventilatory insufficiency, regardless of source, is reflected either in a diminished vital capacity or in a decreased rate of pulmonary air flow of which the 0.5 second expiratory capacity is a measure. This system, therefore, provides a graphic representation of the nature and degree of a

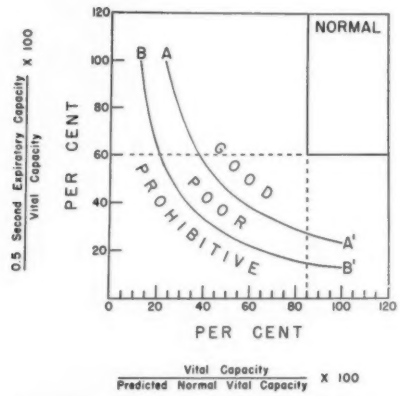

Fig. 1.—Ventilatory function testing system of Miller which provides graphic delineation of pulmonary risk. Patients whose functional plot falls between lines *AA'* and *BB'* are poor pulmonary risks; those whose functional plot is above line *AA'* are good risks; and those below *BB'* are prohibitive risks. Limits of normal for the 2 ratios depicted as coordinates have been established. If an individual cannot expire 60 per cent of his determined vital capacity in 0.5 second, he has a ventilatory defect of the *obstructive* variety. If an individual has a vital capacity of less than 85 per cent of his predicted normal, he has a ventilatory defect of the *restrictive* variety. (Modified after Miller.[6])

patient's ventilatory inadequacy. Fig. 1 shows Miller's method of plotting and delineates the limits between good and poor, and between poor and prohibitive pulmonary risk. Miller derived these limits in a study of 24 patients. Those whose functional plot lay above line *AA'* did not develop major pulmonary complications whereas those whose plot fell between line *AA'* and *BB'* developed such complications. Patients whose plot fell below line *BB'* were considered prohibitive pulmonary risks and on the basis of more extensive pulmonary evaluation were denied operation.[6]

*Warren E. Collins Company, 555 Huntington Avenue, Boston, Mass.

We have employed this graphic method as a guide to surgical planning, and as an indication for pulmonary therapy. All patients outside the *normal* quadrant in Fig. 1 as well as those with a positive cough analysis are placed on the following therapeutic regimen.

Regimen for Improving Respiratory Function Preoperatively.—

a. Cigarette smoking is stopped completely.

b. A wetting agent (Alevaire, 2 c.c. four times a day) and a bronchodilator (Isuprel, 0.5 c.c. four times a day) are administered. These can be given to most patients by means of a nebulizer attached to an ordinary oxygen mask such as the O.E.M. unit.[5] In patients with severe ventilatory insufficiency, however, an intermittent positive pressure respiratory assistor (IPPB) is necessary to insure adequate delivery of the aerosol as shown by Wu.[9] If the mask-nebulizer unit is used, the expiratory-inspiratory-pause method should be employed. With this method the patient expires, then slowly inspires, and pauses before the next expiration.[5]

c. An attempt is made to control specific respiratory infection by obtaining sputum cultures, determining antibiotic sensitivity, and administering appropriate antibiotics.

d. Clearing of secretions is promoted by expectorants (for example, syrup of hydriodic acid) and postural drainage. A mucolytic enzyme, pancreatic dornase (Dornavac, 50,000 to 100,000 units), is used to liquefy tenacious secretions. This is administered by nebulizer each morning.

e. Patients are trained to cough effectively and to make full use of the diaphragm and intercostal muscles in breathing.

f. Patients with bronchospasm are given an aminophylline suppository (500 mg.) on the night prior to and on the morning of operation.

g. Respiratory toilet is encouraged on the morning of operation. The patient uses the IPPB respirator with medications, walks at least 100 feet, and coughs vigorously before receiving preanesthetic medication. If the patient has hitherto required several hours in the morning to clear the overnight accumulation of secretions, operation is scheduled for late in the morning or early in the afternoon.

CLINICAL MATERIAL

During the 6-month period since the initiation of this program at the West Roxbury Veterans Hospital, 250 patients were scheduled for major surgery. These patients were all men. All categories of physical status were represented (Table I). The anesthetics given and type of operation performed may be seen in Table II. Preanesthetic medication was sufficient only to insure psychic sedation. Narcotics were avoided if possible. Secobarbital and scopolamine were the drugs most commonly used. Postoperatively all patients were placed on a stir-up regimen and given only minimal amounts of analgesic drugs. The diagnosis of atelectasis or pneumonitis was based on a temperature over 101° F., together with x-ray or physical findings consistent with decreased aeration or consolidation.

TABLE I. AGE AND PHYSICAL STATUS OF THE 250 PATIENTS EVALUATED*

AGE GROUP	PHYSICAL STATUS			
	1	2	3	4, 5, 6
20-29	32	4		1
30-39	41	9		5
40-49	22	10	2	3
50-59	17	21	2	8
60-69	9	42	12	2
70-79		5	1	2
Total	121	91	17	21

*Graded according to Saklad.[7]

TABLE II. ANESTHETICS GIVEN TO AND OPERATIONS PERFORMED ON PATIENTS EVALUATED

Anesthetics.—	
Spinal or epidural	84
Nitrous oxide, thiopental, curare, alphaprodine	74
Nitrous oxide, thiopental, curare, cyclopropane	55
Cyclopropane, ether, curare	25
Spinal or epidural, and general anesthesia	9
Hypothermia and general anesthesia	1
Total	248
Operations.—	
Lower extremity and hernia, perineal	110
Upper abdominal (stomach and gall bladder)	41
Head and neck, spinal column	34
Lower abdominal (colon and prostate)	21
Kidney and lumbar sympathetic	18
Thoracic	16
Abdominal vascular surgery	8
Total	248

RESULTS

All 250 of these patients received the screening evaluation previously described. Forty-eight (19 per cent) of these patients had a sufficiently suspicious screening evaluation to warrant quantitative testing. Forty of the 48 patients so evaluated had significant ventilatory defects and were placed on the therapeutic regimen. The duration of therapy varied from 3 to 21 days. Improvement in induced cough, ventilatory function studies, or both was noted in 35 of the 40 patients (Cases 1 and 2 are examples). Five patients showed no improvement. During this period 2 patients were refused elective surgery for benign disease because pulmonary function even after therapy continued to be poor. Only 2 of the 250 patients developed postoperative atelectasis or pneumonia. One of these 2 died from extensive bronchopneumonia following an esophagectomy.

CASE REPORTS

CASE 1.—D. G. (W.R.V.A.H. No. 22250), a 66-year-old man, entered for evaluation of a right upper lobe pulmonary lesion. The patient had smoked 30 cigarettes a day for 25 years and had a cough for that length of time. The anteroposterior diameter of the chest was increased, but expansion was normal. Wheezes were audible bilaterally. Chest film suggested emphysema and showed increased bronchial markings at the bases. Induced cough was 2 plus self-propagating and 1 plus productive. The initial V.C. was 2,900 c.c. (90 per cent of predicted normal); the initial 0.5 sec. E.C. was 875 c.c. (30 per cent of determined vital capacity). After 8 days on the therapeutic regimen, the V.C. rose to 3,100 c.c. (98 per cent),

and the 0.5 sec. E.C. to 1,350 c.c. (43 per cent) (Fig. 2). The induced cough became normal. The patient then underwent a wedge excision of the lung for a bronchogenic adenocarcinoma with nodal metastases. The postoperative course was without pulmonary complications.

CASE 2.—H. R. (W.R.V.A.H. No. 22099), a 54-year-old man, was a candidate for mitral valvuloplasty. Left heart catheterization revealed a gradient of 20 mm. Hg across the mitral valve and a calculated valve area of 0.43 sq. cm. However, the patient had a history of recurrent bronchitis and bronchopneumonia and a past history of asthma. He smoked one package of cigarettes a day. Induced cough was 2 plus self-propagating and 3 plus productive. He had a vital capacity of 3,000 c.c. (88 per cent) and a 0.5 sec. E.C. of 1,000 c.c. (33 per cent). With no change in the cardiac regimen of digitalis, bed rest, low-sodium diet, and mercurial diuretics, he was placed on the pulmonary regimen. Within 4 days the cough ceased to be self-propagating and became only 1 plus productive. Vital capacity had risen slightly to 3,150 c.c. (92 per cent), but 0.5 sec. E.C. had risen to 1,775 c.c. (59 per cent) (Fig. 2). The patient underwent valvuloplasty successfully. There were no postoperative pulmonary complications.

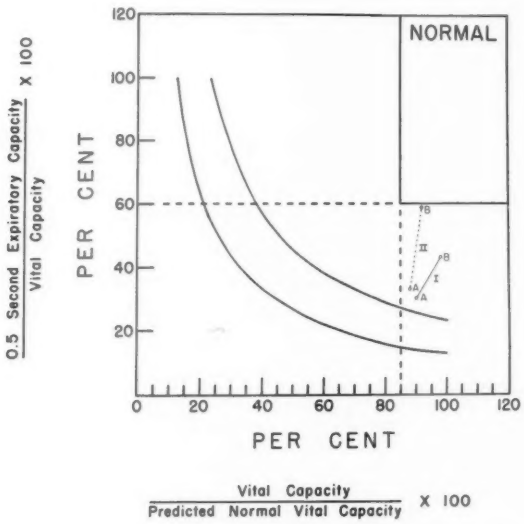

Fig. 2.—Lines *I* and *II* refer to case reports 1 and 2, respectively. Points *A* are before therapy; points *B* are after therapy.

Comment.—In both these patients the improvement in ventilatory function was apparent. We believe the patients' chances of developing postoperative pulmonary complications were decreased accordingly. In the first case ventilatory insufficiency stemmed from bronchopulmonary disease alone. The second case was more complex because the ventilatory defect may have arisen both from cardiac and from intrinsic bronchopulmonary disease. Since the patient was on a constant cardiac regimen, however, the functional improvement and the presumed resultant decrease in pulmonary risk arose largely from an improvement in bronchopulmonary disease.

DISCUSSION

We have used the cough analysis and the ventilatory function testing system with satisfaction as indications for application of a regimen of preoperative pulmonary therapy. Because of an active therapeutic program and a low incidence of complications, we have not attempted to correlate preoperative

evaluation with postoperative complications in treated and untreated patients. Greene[3] has demonstrated, however, that such complications are nine times as common in patients with abnormal induced coughs as in those without. A similar correlation between the quantitative evaluation of pulmonary ventilation and pulmonary risk has been established by the previously outlined work of Miller[6] and by others.[8] We believe these correlations to be reasonable since the prime etiologic factors leading to pulmonary complications are (1) bronchial hypersecretion usually resulting from the irritation of tobacco or infection, and (2) the inability to clear secretions by coughing. Cough analysis is a direct measure of the first factor; the quantitative evaluation reflects both factors since the 0.5 second expiratory capacity is a measure of the obstructive effect of secretions and of the ability to produce a rapid voluminous expiratory effort, that is, a cough.

By providing a good measure of relative pulmonary risk preoperatively, this regimen of evaluation serves three purposes: First, it adds a quantitative value to the many clinical impressions that the surgeon and internist must weigh in deciding for or against operation in every patient. Second, it allows for the selection of patients whose pulmonary vulnerability is great enough to justify careful preoperative respiratory preparation. Time thus spent is akin to time spent in the restoration of cardiac compensation or in the preparation of the bowel prior to surgery. Third, by repeating the evaluation at intervals, the patient's progress can be followed. Serial evaluation provides a measure of improvement in respiratory function and accordingly reflects the resultant diminution in pulmonary operative risk.

Although the group of patients presented is not sufficient to demonstrate the value of this program on a statistical basis, it does point out the prevalence of significant pulmonary risk in a group of general surgical patients. Furthermore, because of the correlation of ventilatory function with pulmonary risk, the results suggest that proper therapy will lower morbidity and mortality.

SUMMARY

1. A plan for preoperative pulmonary evaluation employing the cough analysis technique of Greene and the ventilatory function testing system of Miller has been presented. This regimen comprises a screening component for all preoperative patients and a quantitative component for patients with positive screening evaluations.

2. A regimen of preoperative pulmonary therapy designed to improve ventilatory function and to lessen the risk of pulmonary complications has been presented.

3. Experience with this program in the preoperative evaluation and treatment of 250 patients has been presented.

We wish to thank Drs. Richard Warren, Leroy D. Vandam, and William F. Miller for their advice and criticism in preparing this manuscript.

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CARCINOMA OF THE BREAST

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NO satisfactory set of principles governing the treatment of carcinoma of the breast exists today. Supraradical, radical, simple mastectomy, radiation, hormonal dependency, and biologic predeterminism are among the controversial matters which bombard the surgeon at every turn. Many of these aspects are touched upon in this paper. It was written as an attempt to resolve some of the confusion which exists concerning malignant tumors of the breast. Responsibility for patients demands better scientific management. Even though the paper is based on statistical analysis, it is not designed merely to add new statistical data; nor is any simple rule-of-thumb proposed. The surgeon must still exert his judgment in each case presented to him.

MATERIAL

Three hundred and fifty-seven cases of breast cancer among private hospital white patients, operated on between the years 1925 and 1954, are discussed in this paper. While more than 40 surgeons participated in the series, over 60 per cent of the operations were performed by two of us (Floyd McRae and W. Perrin Nicolson, Jr.). All but a few of the patients were residents of the Atlanta area.

The cases are reviewed as to age and sex of the patient, location and time of discovery of the lesion, the influence of heredity and childbearing, the incidence of metastases, the type of therapeutic procedure, and the relation of these factors to the survival of the patient.

Age and Sex Distribution.—

Despite the relatively small size of the series, the age distribution of the patients is very similar to that of 9,649 cases from the Mayo Clinic,[3] 1,034 from the University of Pennsylvania Hospital,[4] 9,299 from the state of Connecticut,[13] and the two Public Health Surveys of Atlanta in 1937[27] and 1947.[6] As might be expected from a series of private patients, the distribution is shifted slightly toward the younger ages. There are slightly more in the 30 to 40 age group, and fewer in the 60 to 70 age group than in the larger surveys.

The youngest patient was aged 23, and the oldest 87; the greatest number were in the 40 to 49 year age group. The distribution is shown in Table I.

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TABLE I. AGE DISTRIBUTION OF 353 CASES OF BREAST CANCER IN WHITE WOMEN (ATLANTA, GA., 1925-1954)

AGE	NUMBER	PER CENT
80-89	8	2.30
70-79	26	7.47
60-69	51	14.66
50-59	91	26.15
40-49	104	29.60
30-39	60	16.38
20-29	5	1.15
Unknown	8	2.30
Totals	353	100

There were 4 cases of breast cancer in male patients. This is 1.12 per cent of the total. The Public Health Survey of 1937[27] showed one case in Atlanta that year (0.31 per cent), while that of 1947[6] showed none. The percentage for the United States in the 1947 survey was 1.054. Southern cities had fewer such cases in 1947 than did Northern or Western cities. Lewison[23] cites 6 series, both American and foreign, in which incidence in the male ranges from 0.9 to 2.2 per cent of all breast cancer.

All of these male patients were over 50 years of age. One survived 3 months, 2 survived 3 years, and one is still living with no evidence of disease 5 years after operation. (In one patient surviving 3 years, both sides were involved.) In each case, radical mastectomy had been performed.

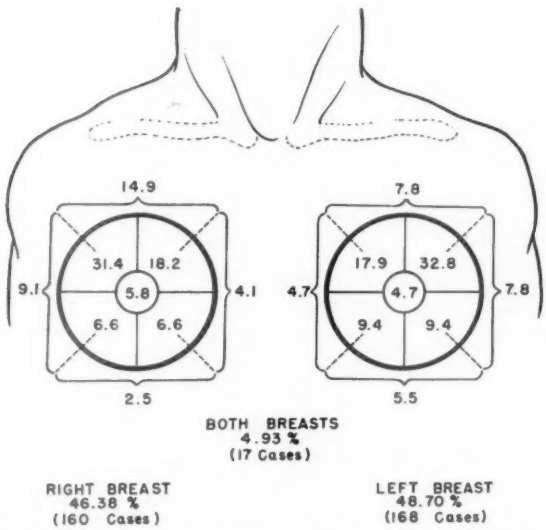


Fig. 1.—Percentage distribution of lesions by location (345 cases). Figures within the outline of the breast refer to the per cent incidence for the 4 quadrants and the center; figures outside the outline refer to the per cent incidence of lesions described as outer half, lower half, and so forth.

Location of the Lesion.—

The most frequent site of the lesion was the upper outer quadrant of the breast, with the upper inner quadrant next. The distribution, shown in Fig. 1, is in essential agreement with Boyd and his associates,[4] Harnett,[14] and most other series. The upper portion of the breast had lesions roughly two and one-half times as often as did the lower portion.

There is a marked difference between the mortality attending lesions in the various portions of the breast. The inner lower quadrant, although the least frequently involved, was the site resulting in the greatest mortality. The outer lower quadrant, with an almost equally low incidence, was the site having the best prognosis. The patients with no evidence of disease 5 years after operation had a higher than average incidence of lesions in this outer lower quadrant. The figures indicate a shift in the mortality rate for the various locations after the age of 50. As mentioned earlier, there is an over-all decline in mortality from the disease after this age. The most notable shift is in lesions of the upper half of the breast. Lesions in this area, which resulted in only 49.5 per cent survival for 5 years after surgery among the younger women, resulted in 74.5 per cent survival among those past the age of 50. In the lower half of the breast, the increase was from 46.4 to 63.1 per cent. The comparison between the two age groups is shown in Fig. 2.

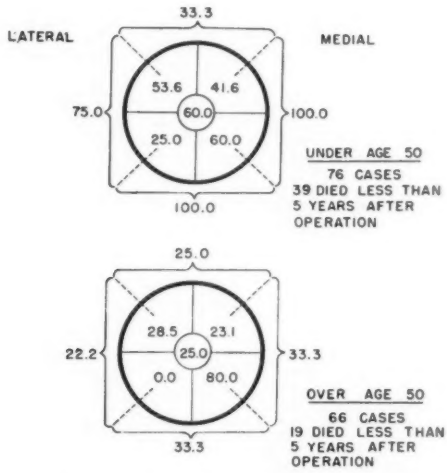


Fig. 2.—The percentage of mortality from lesions in different locations showing the change occurring in patients operated upon before and after age 50, who died within 5 years after operation. (Schema as in Fig. 1, but values for both breasts combined.)

There was no indication that the distribution of the lesion in patients with axillary metastases (the only single metastatic site with enough cases for evaluation) differed at all from that of patients with metastases elsewhere. Fig. 3 shows this comparison.

In the present series, there were slightly more lesions in the left breast than in the right (105:100). Strangely enough, the patients operated upon before age 50 had fewer lesions on the left (92:100) while the older patients had many more (120:100). There was no evidence of a difference in the survival rate between lesions of the two sides.

Busk and Clemmeson[5] in their Danish series showed a greater difference between left and right lesions (111:100). This same difference was found in England by Harnett[14] and among nonwhite women in the United States by Dorn and Cutler.[11] White women in the United States had slightly fewer lesions on the left than nonwhites (108:100).

No satisfactory explanation has been offered yet for this higher incidence of involvement of the left breast. When trauma was still considered an etiologic

factor in breast cancer, Lane-Claypon[20] collected evidence that injury occurred more often to the left than to the right breast by a ratio of 92:60. Unfortunately for this argument, trauma may lead to discovery of a tumor, but can no longer be considered to cause one.[19] Only 14 of the patients in the present series reported antecedent trauma to the breast (presumably to the one affected).

Involvement of both breasts was found in 17 patients (4.94 per cent) of the series. Harrington[15] reported 3.2 per cent, Desaive[9] found 8.02 per cent, while Harnett found only 0.3 per cent. The actual figure in any series depends upon the duration of the follow-up available.

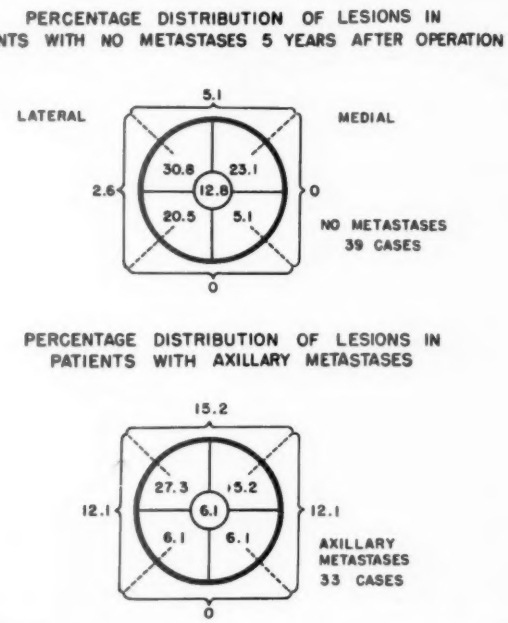


Fig. 3.—The percentage of distribution of lesions in patients with no metastases and in patients with axillary metastases 5 years after operation. (Schema as in Fig. 1, but values for both breasts combined.)

Discovery of the Lesion.—

The presenting complaint was pain in 20 cases of this series, nipple retraction in 8, and the presence of a lump in 65 cases. Trauma was alleged in 14 cases (7.95 per cent) and nipple discharge was present in 19 (10.80 per cent) in cases where records were available. The remainder were unstated.

Of the 293 cases in which time of first discovery by the patient could be obtained, 41.6 per cent were operated on within one month, and 90.8 per cent within one year (Table II). The longest delay was 18 years. This is a marked contrast to the figures for a series of 905 charity patients from Atlanta (1924-1942) presented in an earlier paper by one of us (Nicholson[28]) in which only 13.9 per cent were treated within 1 month, and only 72.9 per cent within the first year.

The relation between survival time and early discovery is not encouraging. Of those patients who died of cancer less than 2 years after operation, 35.1 per

TABLE II. TIME FROM PATIENT'S DISCOVERY OF THE LESION TO OPERATION IN RELATION TO OUTCOME OF OPERATION

NUMBER OF CASES	PER CENT OF TOTAL	TIME OF TREATMENT AFTER DISCOVERY (%)		OUTCOME OF OPERATION
		1ST MONTH	1ST YEAR	
37	12.6	35.1	91.9	Died of cancer under 2 years
45	15.4	51.1	91.1	Died of cancer over 2 years
52	17.7	32.7	92.3	Alive 5 + years, free of disease
40	13.7	55.0	87.5	Alive 5 + years not free of disease
119	40.7	39.5	90.8	Alive with less than 5 years of record
293	100.0	41.6	90.8	Totals

cent had been operated upon within one month of discovery of the tumor. Of those who died more than 2 years later, 51.1 per cent were operated upon within one month. The difference is, however, not very significant (about a 20 per cent probability that the difference is due to chance: $p = 0.20$) and is further weakened by the fact that only 32.7 per cent of those alive with no evidence of disease 5 years later were treated in the first month. Of those treated in the first month, living over 5 years but not free from disease, the percentage was 55.0 per cent.

Among those who eventually died of their disease and had been treated within the first month, 43.9 per cent (36 cases) lived an average of 40.3 months after the operation. Of those treated over one month but less than 12 months after discovery, 36.6 per cent (30 cases) lived 31.7 months. Of those in whom delay was more than one year, 19.5 per cent (16 cases) lived an average of 34.0 months. These differences are barely significant. Table II summarizes time of patient's discovery prior to operation with relation to eventual outcome.

Time elapsing from discovery by the patient to treatment by the surgeon has not appreciably decreased over the period of years covered by this study. Excluding 5 extreme cases in which the disease was said to have existed for more than 5 years prior to operation, the mean time from the patient's discovery to operation is shown in Table III.

TABLE III. MEAN TIME FROM PATIENT'S DISCOVERY OF LESION TO OPERATION BY 5-YEAR PERIODS

PERIOD	NUMBER OF CASES	MEAN TIME DISCOVERY TO OPERATION (MONTHS)	NUMBER OF CASES EXCLUDED FROM MEAN
1935-1939	21	6.81	
1940-1944	51	4.72	(1 over 5 years)
1945-1949	91	7.57	(1 over 5 years)
1950-1954	120	6.87	(3 over 5 years)
1935-1954	283	6.70	5

Heredity.—

Incidence of cancer in the patients' families was determined for 152 cases. Of these, 65.1 per cent reported no cancer in the family, 26.3 reported one

parent or sibling as having cancer, and 8.6 per cent reported 2 or more. There was no difference between those who lived a longer or a shorter time, or who seemed cured. Table IV shows the percentage in each group.

TABLE IV. EVIDENCE OF CANCER IN FAMILIES OF 152 PATIENTS OPERATED UPON FOR CANCER OF THE BREAST

PATIENTS	FAMILY HISTORY OF CANCER	
	NEGATIVE (%)	POSITIVE (%)
Lived over 5 years	62.7	37.3
Lived less than 5 years	67.7	32.3
Died of cancer	65.8	34.2
Free of disease 5 years	65.2	34.8

This rate of almost 35 per cent with positive family history is much higher than the figures usually given. Lewison and Allen[22] report 25.3 per cent, and the earlier study of charity patients in Atlanta[28] reported only 12.2 per cent. (It is likely that this latter figure is too low due both to ignorance of the patient about her family history and to confusion with euphemisms for cancer.) Even Lewison and Allen's noncancerous control group reported a rate of 14 per cent cancer in the family.

Why the rate in this series should be so far above the expected rate is another matter. Without confirmation, it seems unreasonable to believe there could be a geographic difference, yet such may well be the case. That the 35 per cent is more than double the expected rate merely re-emphasizes the by now well-established conclusion (Jacobsen[16]) that hereditary predisposition to breast cancer exists.

Influence of Childbearing.—

In 189 patients of the series, there was information on the number of children and miscarriages. One or more live children were born to 113 patients, 9 more had had miscarriages only, and 67 reported no pregnancies. It was not possible to divide this latter group into married or unmarried women.

Of these 189, 58 died of breast cancer. Forty-two (72.4 per cent) of those who died had had one or more pregnancies prior to operation. Of the 37 women who were free of disease 5 years later, only 18 (48.6 per cent) had ever been pregnant.

Of all women who lived more than 5 years after operation, including both those considered cured and those who eventually died of the disease, 52.1 per cent (37) had borne children and 47.9 per cent (34) had not. In that group that died less than 5 years after operation, 32 (71.1 per cent) had borne children, and 13 had not. Restated in other terms: of all who had children, 53.6 lived over 5 years after operation; of all who had *no* children, 72.3 lived over 5 years. These differences between the reproductive history of patients living more than 5 years and less than 5 years after operation are statistically significant (p is less than 0.001, or one chance in 1,000 that the difference is accidental). Actual numbers and percentages of pregnancies in different groups of patients are shown in Table V.

TABLE V. EFFECT OF PREGNANCY AND CHILDBEARING ON POSTOPERATIVE RESULTS IN CANCER OF THE BREAST

NUMBER HAVING CHILDREN	NUMBER HAVING ONLY MISCARRIAGE	NUMBER HAVING NO CHILDREN	PER CENT EVER PREGNANT	OUTCOME
39	3	16	72.4	Died of cancer
5	0	3	62.5	Died, unknown cause
18	0	19	48.6	Lived 5 years, free of disease
12	1	12	48.0	Lived 5 years, not free of disease
39	5	17	72.2	Alive with less than 5 years of record
37	-	34	52.1	Total lived over 5 years
32	-	13	71.1	Total lived under 5 years

The incidence of death from breast cancer among the unmarried had been shown by Dorn[10] and others to be higher than among the married, and, of the married, higher among the childless than among those with children. Within the childbearing period, however, the present series shows that women who have borne children are more vulnerable than women who have not.

Logan[24] also observed that " . . . childbearing, or possibly the marital state itself seems to predispose to death from cancer of the breast." The incidence among the childless is only slightly higher in the present series, but the progress of the disease seems to be slower among them than among those who have borne children. (It is interesting to note that those free of disease 5 years after operation had an average of 2.06 children per mother, while those similarly alive, but with metastases, had 2.77 children per mother.) Thus, while more childless women may die of breast cancer, it would appear that its onset is later in life and its progress is slower than in mothers.

Incidence of Metastases.—

Metastases occurred in 87.2 per cent of all who died from any cause and in 94.3 per cent of all who definitely died of cancer. The rate for those alive over 5 years but not free of disease was 34.9 per cent, while, for those whose records have not yet run for 5 years, metastases were found in only 27.9 per cent. To some extent, this last figure simply reflects the lack of time for metastases to manifest themselves.

Table VI shows the distribution of metastases. Sites of metastasis were those usually found, with the following exceptions: One patient, aged 68, whose lesion was first found 8 weeks prior to operation, had metastasis to liver 26 months and to cecum 36 months postoperatively; one patient, aged 53, had metastasis to uterus 29 months postoperatively; one patient, aged 59, of 3 months' preoperative duration, developed abdominal ascites 7 months after operation. All 3 had undergone radical mastectomy.

Irradiation and Survival.—

One hundred and thirty-two of the patients having a 5-year record were given x-ray therapy following their operations. This amounted to 67.7 per cent

TABLE VI. METASTATIC SITES AND OUTCOME OF OPERATION

OUTCOME	NO METASTASIS REPORTED	UNSPECIFIED METASTASIS	AXILLA	BONE	LUNG	OTHER*	PER CENT WITH GENERALIZED METASTASIS	
Died	15	11	13	25	11	27	23	88.0
Alive over 5 years but not free of cancer	25	0	7	2	3	3	0	37.5
Alive less than 5 years of record	91	2	20	3	2	8	1	28.3
Alive over 5 years free of cancer	55	0	0	0	0	0	0	0.0
Totals	186	13	40	30	16	38	24	46.4

*Other Sites:

Brain only	8	Cases fewer than 3	
Brain and other	6		Skin
Liver only	3		Larynx
Liver and other	3		Trachea
Lung and other	7		Mediastinum
Supraclavicular nodes	5		Cecum
			Uterus

of the cases in which information was available. Among the patients undergoing radical mastectomy, more of the longer-lived group received radiation than of the shorter-lived group. Most of the patients subjected to simple mastectomy who died less than 5 years later also had radiation therapy. About one-half of the longer-lived group were so treated. These results do not permit positive conclusions. The greater frequency of radiation in short-lived patients of the simple mastectomy group probably represents palliative treatment of individuals considered poor risks for a radical operation or patients whose disease had already become inoperable. Detailed figures are shown in Table VII.

TABLE VII. IRRADIATION

	TOTAL NUMBER OF CASES	CASES IRRADIATED		
		NUMBER	PER CENT	OUTCOME
Simple mastectomy	29	14	48.3	Lived over 5 years
Simple mastectomy	14	12	85.7	Lived less than 5 years
Totals	43	26	60.5	
Radical mastectomy	79	58	73.4	Lived over 5 years
Radical mastectomy	73	48	65.8	Lived less than 5 years
Totals	152	106	69.7	

Survival Rates.—

Rate of survival was calculated by the method described by Berkson and Gage,[2] and by the Connecticut survey.[13] It is especially adapted to series such as the present one in which some patients have been lost to follow-up at varying lengths of time after operation, and others had not died at the time the series was terminated.

To eliminate all cases not followed completely is to needlessly reduce the number available. To include them involves one of three assumptions: (1) all who are lost to follow-up have died, (2) all who are lost to follow-up have recovered and are alive, and (3) those who are lost to follow-up have lived or died in the same proportions as those actually followed. The truth must, of

course, lie between *1* and *2*; these assumptions represent the worst possible and the best possible survival rates. Choice *3* is the most reasonable estimate of the probable situation and is the one obtained by the Berkson and Gage method.

Crude survival curves do not tell the whole story. Over the years, patients are subject to death from other causes than the disease for which they were operated upon. A woman of 65 does not have the same 15-year survival chances as does one of 30, no matter how effective the surgery. A survival curve should be compared with the normal expected 5-, 10-, and 15-year survival values for a group of white women of the same age composition as the group of cancer patients under study. Such values may be considered as a "control." (For this series, these values are based upon the 1949-1951 Life Tables for the United States.[30]) It is not a perfect control in that it is national, not local; it includes deaths from cancer of the breast along with all other causes of death; it is for a specific 2-year period, not for the whole 22 years represented by the patients in the present series.

Survival Rate and Age.—

While there are too few patients for analysis in the oldest and youngest age groups, between the ages of 30 and 80, the fewest number of 5-year survivals was found among the patients 40 to 49 years old at the time of operation. Fewer than one-half (47.7 per cent) of these patients survived 5 years.

In contrast, the next older group, those from 50 to 59, showed a much better response, with 65.5 per cent surviving 5 years. Taking all patients over age 50 and comparing them with all under 50, the 5-year survival rate is 62.5 per cent for the older, and 51.0 per cent for the younger group.

TABLE VIII. AGE AND SURVIVAL

AGE GROUP	NUMBER OF CASES	SURVIVAL			
		5 YEARS		10 YEARS	
		NUMBER	PER CENT	NUMBER	PER CENT
80-89	1	0	0	0	0
70-79	12	7	58.3	2	16.7
60-69	28	17	60.7	2	7.1
50-59	55	36	65.5	17	30.9
40-49	63	30	47.6	11	17.4
30-39	38	22	57.9	5	13.1
20-29	3	1	—	1	—
Over 50	96	60	62.5	21	21.9
Under 50	104	53	51.0	17	16.3

Of those 40- to 49-year-old patients who lived for 5 years, 36.6 per cent were still alive 10 years after operation. Of the 50- to 59-year-old group, 47.2 per cent were still alive, even though the normal female population aged 50 to 59 has only 92 per cent of the 10-year expected survival of the younger group. The 5- and 10-year survivals are shown in Table VIII. (Those lost to follow-up or alive but operated upon less than 5 years ago are not included.)

Considering those who have survived 5 years *with no recurrence* of their disease, there is a less marked difference between those over 50 years of age and

those under, although the older patients still show better results. This was not the case in the 1924-1942 study of charity patients in Atlanta.[28] In that group, more of the younger patients survived 5 years than did the older ones. The differences in survival time between the younger and older patients is not of great statistical significance, and many other observers have not reported it. In the present series, there is about a 10 per cent probability that the difference is due to chance (p is equal to 0.10). Nevertheless, its association with the period of menopause and the cessation of ovarian function suggests that there is a real relation between rate of tumor growth and lowered ovarian hormone levels. The fact that the effect is more pronounced among patients with metastases than among those in whom all signs of cancer appear to have been eradicated incline one more strongly to this belief.

Anderson and his associates[1] have shown that there is a slight decrease in the number of new cases of breast cancer arising in the immediate postmenopausal years. They found this drop in incidence in a number of series in the United States, England, and Denmark. They do not mention the change in mortality which was observed in the present series and, on the other hand, their postmenopausal decrease in incidence did not appear in our data.

The suggestion has been made by Jessiman and Moore[17] that breast tumors may be classified as "estrogen-stimulated," "pituitary-stimulated," and "hormone-independent." They feel that most of the tumors beginning before the menopause are of the "estrogen-stimulated" type, while those occurring in the senile woman must be "hormone-independent." They further cite evidence that while urine estrogen declines after menopause or therapeutic castration in most women, in some, stromal hyperplasia or adrenocortical activity 4 to 6 years later may raise urine estrogen above the premenopausal level. In such patients, there is a decrease in the rate of tumor growth after menopause, followed by a return to the earlier rapid spread. Finally, these authors believe that in senility the hormone-stimulated tumor may become "hormone-independent."

The slight decrease in postmenopausal mortality found in the series reported in this paper is accounted for by the Jessiman and Moore theory. It also explains why the decrease is not more marked.

Survival Rate and Type of Operation.—

In Figs. 4 and 5, the survival rates following simple and radical mastectomies are shown. The solid line represents the assumption that patients lost to follow-up died at the same rate as the patients actually followed. The two broken lines indicate the two extremes of possibilities that all lost have died or that all lost are still living.

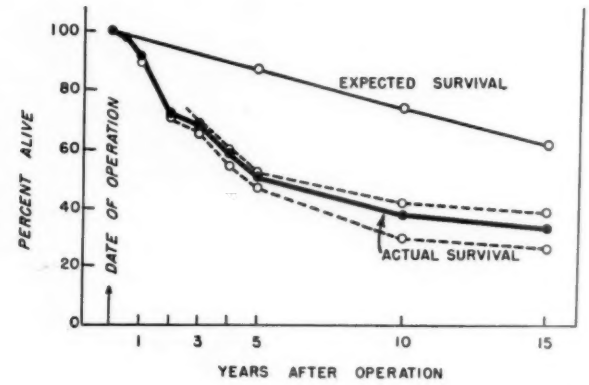
The 5-year survival rate following radical mastectomy was 57.2 ± 3.4 per cent.* This is less than two standard errors above the 53.2 per cent survival of the large Mayo Clinic series and is not significant. The 10-year survival rate of 47.7 ± 3.6 per cent lies between two and three standard errors above the 38.1 per cent of the Mayo series. As the survival rates for the latter series has risen continuously over the 24 years covered, comparisons are difficult.

*Standard error of estimate, by method of Cutler.[6]

Effectiveness of Surgeons.—

Meyer and Smith[26] made a comparison between the 5-year survival rate obtained by 5 surgical specialists (49 per cent) and 22 "infrequent operators" (50 per cent). They concluded that "neither the technical proficiency of the operators nor the extent of the operation had any effect on the 5-year survival rate."

Fig. 4.



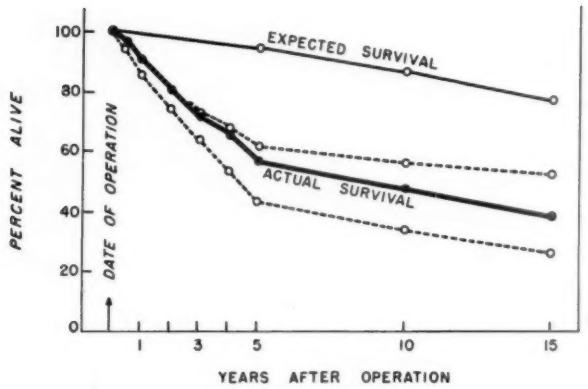


Fig. 5.

Fig. 4.—Survival rate of patients undergoing simple mastectomy. Upper curve represents expected survival rate of the general U. S. population of the same age composition as the patients. Dotted lines represent upper and lower limits of possible survival rate as explained in text.

Fig. 5.—The survival rate of patients undergoing radical mastectomy. (Details as in Fig. 4.)

In the present series, a similar division has been made of the results obtained by surgical specialists who performed 140 operations for which 5-year survival rates could be calculated and occasional operators who performed 64 such operations. The results do not agree with those reported by Meyer and Smith. The best qualified surgeons with the greatest experience obtained 64.3 per cent survival after 5 years, while the infrequent operators obtained only 40.6 per cent survival. Table IX shows these results by type of surgical procedure. It is obvious that experience does make a difference in results obtained. It is interesting to note in passing that the specialists performed simple mastectomy in almost one-third of their patients, while the occasional operators rarely employed it.

TABLE IX. 5-YEAR SURVIVAL RATES OF BREAST CANCER PATIENTS OF TWO GROUPS OF SURGEONS

	SURGICAL SPECIALISTS	INFREQUENT OPERATORS
Number of patients		
Radical mastectomy	98	55
Simple mastectomy	38	3
Other procedures (including no operation)	4	6
Total	140	64
Lived less than 5 years		
Radical mastectomy	38	32
Simple mastectomy	11	2
Other	1	4
Total	50	38
Lived more than 5 years		
Radical mastectomy	60	23
Simple mastectomy	27	1
Other	3	2
Total	90*	26†
Per cent survival		
Radical	61.2%	41.8%
Simple	71.1%	33.3%
Other	75.0%	33.3%
Total	64.3%	40.6%

*Of these, 46 (51.1 per cent) showed no evidence of disease 5 years after operation.
†Of these, 9 (34.6 per cent) showed no evidence of disease 5 years after operation.

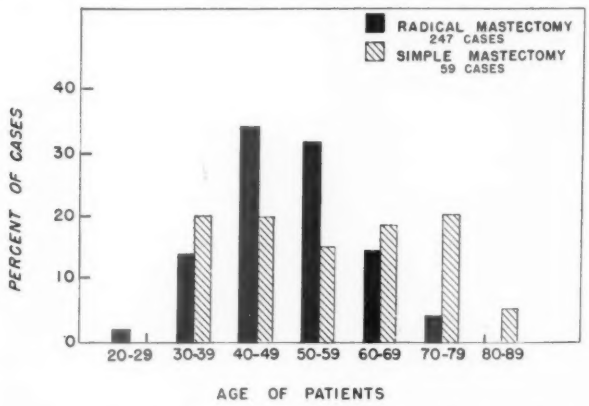


Fig. 6.—Percentage distribution of patients by age at operation. A comparison of radical and simple mastectomy (306 cases). The additional patients in the series underwent simple excision or were not operated upon.

DISCUSSION

Comparison of Results of Simple and Radical Mastectomy.—

It is obvious from Figs. 4 and 5, that the crude survival rate for patients undergoing simple mastectomy is lower than for those undergoing radical mastectomy. Five- and 10-year crude survival rates are not, however, enough for an evaluation. Because of the difference in life expectancy at different ages, the age distribution of the patients must be considered. This distribution in the two groups is very different. Fig. 6 shows the comparative percentage distribution by decades.

When the actual survival rates of the two groups are expressed as percentage of the expected survival of populations of similar age distribution, the radical

procedure appears to have been very slightly more successful at 5 and 10 years after operation. At 15 years, a larger percentage of those who would otherwise have been expected to live for 15 years survived following simple mastectomy. Table X shows the results of corrected survival rates.

TABLE X

PER HUNDRED PERSONS	NUMBER ALIVE		
	AFTER 5 YEARS	AFTER 10 YEARS	AFTER 15 YEARS
A. Survival Following Simple Mastectomy			
General female population (same age composition as patients) expected to be alive at end of period (from U. S. Life Tables for 1949-1951[30])	87.21	74.16	61.99
Cancer patients actually surviving operation	50.37	38.08	33.95
Cancer patients expected to survive untreated (from Daland[7])	22.0	5.0	0
Cancer patients actual survival as per cent of expected survival	57.76	51.35	54.77
B. Survival Following Radical Mastectomy			
General female population (same age composition as patients) expected to be alive at end of period	94.54	87.15	77.82
Cancer patients actually surviving operation	57.24	47.70	38.82
Cancer patients expected to survive untreated	22.0	5.0	0
Cancer patients actual survival as per cent of expected survival	60.55	54.73	49.88

If the procedure is reversed and those patients with a complete record (120) are divided into those who lived 5 years or more and those who failed to do so, we find that 25.9 per cent of the former and only 15.7 of the latter underwent simple mastectomy. These figures seem to favor simple mastectomy. The group with the largest percentage (27.3 per cent) of simple mastectomies was that in which the patients showed no evidence of disease 5 years after operation.

The best response is shown by this group without metastases at operation and with the slowest growing tumors (or perhaps the shortest preoperative delay, although the data here will not confirm this). These tumors would have responded equally well to radical mastectomy and would probably have shown the best survival time, even in the absence of surgical intervention.

In their Rockford, Illinois, series, Meyer and Smith[26] have concluded that the extent of the operation had no effect on the 5-year survival rate.

Deaton[8] has collected a number of series to make a similar comparison. Without correcting for the age distribution or the normal life expectancy of the patients included, he reports a 5-year survival rate of 59.8 per cent for 808 patients undergoing simple mastectomy and 54.5 per cent for 2,955 undergoing radical mastectomy. He emphasizes the point that any alternative

which avoids the mutilation of radical mastectomy, and yet which will have as good results measured by survival rate, should be extremely attractive to the surgeon.

It is recognized that with the greatest care in the world there will be some patients in the simple mastectomy group operated upon because of the bias *toward,* not away from, diagnosis of malignancy. Lees and Lees[21] have stated what many others, no doubt, have felt but have not said:

> There is not only the obvious fact that a non-malignant tumor wrongly diagnosed as malignant becomes a '5 year cure' while a malignant tumor wrongly diagnosed as non-malignant soon has its diagnosis changed. . . . It . . . is much more difficult and responsible to diagnose non-malignancy rather than malignancy in a doubtful case.

Effectiveness of Treatment.—

Recently, the Edinburgh group has questioned the effectiveness of all types of treatment for breast cancer. Lees and Lees[21] have suggested that, if the classical viewpoint of Halsted be correct, one must be able to show that delay in treatment diminishes the cure rate. These authors contend that there is very little evidence from the published literature that this *is* the case.

Park and Lees,[29] in an extensive survey of reported series, criticize a number of older reports seeming to show that there is a good correlation between preoperative duration and survival time.

The figures in the present series tend to bear out the contention of the Edinburgh group. Correlation between time from patient's discovery to operation and time from operation to death is very poor indeed (coefficient of correlation equals 0.02). Fig. 7 is a scatter diagram of the relation between these two intervals of time. It is plotted on a logarithmic scale in order to include a few extreme cases without unduly obscuring the shorter-lived cases. Only those known to have died from their disease are included in order to eliminate any possible spurious "5-year cures." The delay from discovery to operation for patients still alive, or dying otherwise, is given in Table II. Manifestly, time of discovery by the patient is a variable which is not under the physician's control and is by no means as accurate as one would wish. Nevertheless, it is the only index available and, by virtue of years of public education, it is probably more accurate for breast cancer than for many other diseases.

It is possible that we may not be dealing with the simple spread of malignant cells through lymph channels; still earlier diagnosis and the more extensive surgery of supraradical operations may not necessarily result in further decrease in mortality or morbidity.

MacDonald[25] has held that the course of neoplastic disease depends upon an interaction between tumor and host cells (biologic predeterminism) and that the growth is not "analogous to the spread of termites in wood." If this be the case, then there may be no strict time-distance relationship, and survival time may not be related to onset time, but to the nature of the tumor

itself. This view has been strongly supported by Gatch,[12] who has contended that improper radiation and extensive surgery may serve to destroy the "body's innate resistance to new growths."

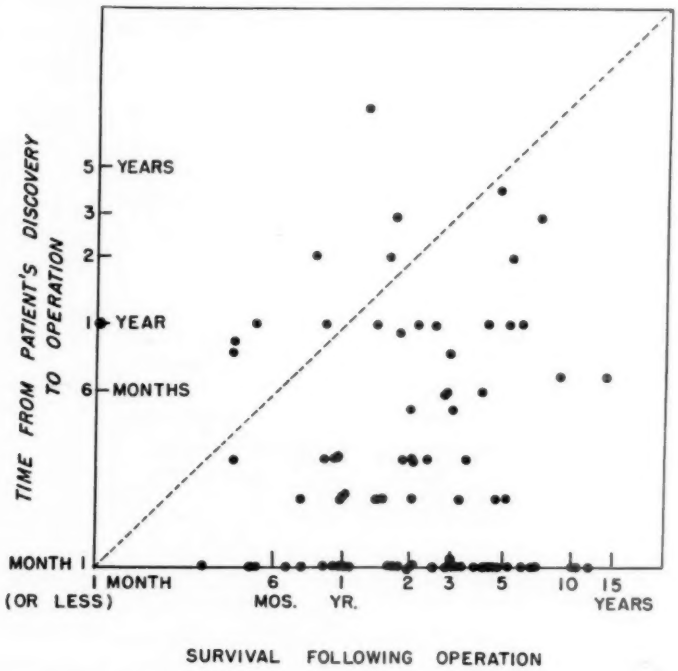


Fig. 7.—Relation between immediacy of operation and time of survival. Logarithmic plot of time elapsing between patient's discovery of the lesion and operation against patient's survival following the operation. Eighty-one patients with known preoperative duration of lesion who have ultimately died of their disease. Points lying to the left of the dotted line represent patients for whom postoperative survival was shorter than preoperative duration of the disease. Logarithmic plot is used in order to include extreme cases without unreasonable crowding of those with shorter survival time.

If, on the other hand, the older view is correct, then the rapidity of the spread of the tumor must be so variable that prognosis is difficult. In 9 patients, operation upon tumors within one month resulted in survival of less than one year, while in 6 other cases, operation a year after discovery resulted in survival from 16 to 72 months. Early discovery may mean only a rapidly growing cancer which will end in death so much the sooner. This is the probable explanation for the fact that, among patients dying of breast cancer, those operated upon within 2 to 11 months of discovery show a shorter mean survival time (31.7 months) than those operated upon immediately (40.3 months) or those operated upon more than 12 months after discovery (34.0 months). Keyes and co-workers[18] have also emphasized the small difference in survival time resulting from "early" and "late" mastectomy, although, their figures were not calculated on the same basis as those of the present series.

In 9 cases, the patients lived longer (average, 28.78 months; maximum, 10 years) between discovery and operation than they did following surgery (9.67 months). Their total life from discovery to death was 38.45 months,

which was a longer total life than 61.1 per cent of those operated upon within the first month, and compares well with the 40.3 months average life of the latter group. If the single case of over 10 years' duration be omitted as an unusual extreme, the average survival was 27.6 months, or as long as 44.4 per cent, of those operated upon within the first month.

This is not to say that at the present state of knowledge the surgeon should delay operation. It is merely to point out that early surgery is not the comfortably easy solution to the problem. Any cancer that has persisted for a year without killing the patient is, by definition, a slowly growing one. Conversely, at the other extreme, the fastest growing cancer is already inoperable at the time of discovery, is soon fatal, and hence often does not become included in surgical series.

Park and Lees[29] further criticize the use of the 5-year survival rates as measures of effective therapy. While their criticism may have some validity, the data, shown in Figs. 4 and 5, indicate that, in this series, the death rate of patients after the first 5 years has almost exactly the same slope as does the general population death rate (upper curve). Thus, barring a few exceptions, one may be justified in saying that patients who have survived 5 years since mastectomy have almost as good a life expectancy as have the general population of women of the same age. The expression "5-year cure," while of little comfort to the individual patient with slowly growing metastases, seems to have some real meaning for patients as a group.

For example, from the figures for radical mastectomy, in Table X, there were 57 patients of 100 operated upon who were alive 5 years after surgery. Five or 6 of the 100 would have died from a variety of causes in this period (1949-1951 U. S. Life Tables[30]). Thirty-seven more actually did die from breast cancer, leaving the 57 survivors. These 37 deaths were not prevented by operation, and we must not minimize this fact. Had the patients not undergone operation at all, however, there would have been only 22 survivors (Daland[7]). Thus, there were "cured" 57 less 22, or, 35 of the 100 patients with radical operations.

In another 10 years (15 years postoperatively), some 16 more patients will be expected to die from all causes, according to the life tables. If the 35 "cures" are permanent, there would be lost from the original 100 patients:

6 expected deaths from all causes in the first 5 years
37 additional deaths from breast cancer in the first 5 years
16 expected deaths from all causes, 5 to 15 years

59 deaths from 100 = 41 survivors.

Thus, theoretically, from 100 patients, 59 would have died, leaving 41 survivors at the end of 15 years. None would have survived untreated, according to Daland.[7] If 5 years' survival constitutes a "cure," 41 patients should be alive after 15 years. In this series, there are actually 38.8 survivors per 100 patients, or 95 per cent of the predicted number. Only slightly more than 2 patients per 100 have died over and above the expected number of

deaths from all causes during the period. It is probable that the record is somewhat poorer than it appears, as the mortality rate for these private patients may be less than the national average.

The foregoing analysis tells us only that, if a patient is going to die of breast cancer, she will, in all probability, do so within 5 years after operation. If she does not die, either we have succeeded in extirpating the malignant cells (Halsted), we have arrested them in the lymph nodes and perhaps destroyed them by irradiation (McWhirter), the patient's own tissue composition has altered so that a predisposition to cancer formation no longer exists (MacDonald), or, finally, the tumor was not malignant to start with!

SUMMARY AND CONCLUSIONS

1. Three hundred and fifty-eight (358) cases of breast cancer in Atlanta, Georgia, from 1925 through 1954 are described. All were private patients.

2. Lesions of the inner lower quadrant of the breast are the least common, but result in the highest mortality. Lesions of the outer lower quadrant of the breast are equally rare, but result in the lowest mortality.

3. A postmenopausal decrease in mortality is observed; it is more marked among patients with lesions of the upper portion of the breast than among those with lesions of the lower portion.

4. A slight predominance of left breast lesions (105:100) was found in this series.

5. The preoperative existence of the lesion was shorter than in a series of charity patients from the same area. Time from patient's discovery to operation has not markedly decreased over the years included in this series. There was a mean improvement of less than 9 months' survival time in the patient operated upon within one month of discovery of the lesion, in comparison with those operated upon between 2 and 12 months of discovery.

6. Positive family history of cancer was 35 per cent. This is higher than the usual figures given and nearly three times that of charity patients in Atlanta.

7. Among patients who have not borne children, breast cancer appears later in life and progresses more slowly than in patients who have been mothers.

8. Five-year survival was better (62.4 per cent) in patients over the age of 50 than in younger patients (51.0 per cent).

9. In contrast with other studies, the present series indicates that specialists who have performed many mastectomies have had appreciably better results than have infrequent operators.

10. Results of simple and radical mastectomy are compared, and their significance discussed. With proper selection of individual cases, the simple procedure seems as good as the radical.

11. Survival rates, preoperative duration of disease, and effectiveness of treatment are discussed with respect to sources of confusion due to differing analyses of data.

12. The need for standardization of treatment is obvious; the hope of achieving it cannot yet be fulfilled.

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IN DEFENSE OF THE CONSERVATIVE TREATMENT FOR ACUTE CHOLECYSTITIS WITH AN EVALUATION OF THE RISK

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THE choice of treatment for acute cholecystitis has and still does provide reason for discussion. As in other conditions such as gastrointestinal hemorrhage there is the choice between immediate operation, as soon as the patient's condition will permit, and conservative treatment. The latter is followed by surgery, if permitted, at a time of election and after diagnostic studies have been done. Immediate operation presumes a correct but frequently unconfirmed diagnosis and accepts the hazards of surgery, not of election. It offers a substantial reduction in hospital stay. Conservative treatment with operation delayed accepts the dangers of gall-bladder perforation or pericholecystic abscess and offers possible operation of election. Which method of treatment offers the best care for the patient is still open to discussion. This statement is particularly true if it is realized that principles of treatment excellent for one institution may be most harmful if applied indiscriminately to other hospitals.

Glenn,[5] following the principles laid down by Heuer, is known as the chief proponent of immediate operation as a hospital service policy. Mulholland and associates,[4] Zollinger and co-workers,[10] and Coller and colleagues[2] may be termed conservatists with the reservation that individual case evaluation is necessary to recognize the failure of conservative treatment. Ochsner and co-workers[3] and Reines and Kessler,[8] after reviewing their experience, urge prompt surgery. From reports of immediate surgery it is difficult to ascertain the percentage of patients that could not safely be readied for immediate surgery. Reports of the conservative treatment indicate a variation in the number of cases requiring emergency surgery for uncontrolled disease, urgent surgery for failure to improve and the number treated by surgery of election. As suggested by Mulholland, this variation could be explained by the difference between "watching" acute cholecystitis and "vigorous conservative treatment." It has been pointed out by Coller and others that the majority of gall-bladder disease is not treated in medical centers. What is the wisest policy to adopt for the average community hospital?

Conservative treatment has been the practice at St. Joseph Mercy Hospital, Ann Arbor, for many years. However, it was only after a review from this hospital in 1950[9] that attention was drawn to proper conservative treatment in this hospital. Although improvement has occurred with proper treatment, that is, gastric suction, intravenous fluids, and sedation instead of

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watching, there are cases still not energetically treated. However, we can say that in this community hospital no patient needing treatment for acute cholecystitis languished or died on the medical service. Surgical consultation was obtained for all problem cases of acute cholecystitis. The results of this policy are presented as a defense for conservative treatment with the hope that organized community hospitals will reconsider this treatment.

During the 5-year period from 1952 through 1956, 752 patients were treated in the hospital for biliary tract disease. Of these 345 (45 per cent) were treated for acute cholecystitis. This high percentage of acute biliary disease is explained by the fact that this is a community hospital serving an area with relatively constant and high economic level where hospital rather than home care is accepted for acute disease. Those patients with adequate history, physical findings, and laboratory work to warrant treatment for acute cholecystitis were considered to have had acute cholecystitis and so are included in this series. Not included are those who were erroneously so diagnosed and later were proved to have had another disease. Not included are those who were treated for another disease and later were shown to have had acute cholecystitis.

None was treated by "immediate, prompt, or early surgery," meaning surgery performed as quickly as the condition of the patient would permit. All operated upon within 72 hours of their admission were treated as of necessity. Therefore, acute cholecystitis was treated conservatively in all patients, meaning operation was delayed if possible until the disease was quiescent and the indicated diagnostic studies were completed. If delay was not possible, surgery was done as an emergency or considered urgent during the course of treatment.

Of the 345 cases of acute cholecystitis, 154 (44.3 per cent) were discharged without operation during this hospitalization. Thirty per cent of these returned for operation at a later date. The reasons for discharge are listed in Table I. The possible impact of immediate operation on this group cannot be stated except that the mortality could not have been less since there were no deaths. Undoubtedly an aggressive attitude toward immediate surgery could have led many of these patients to operation within 48 or 72 hours. Assuming that our clinical diagnosis was correct in all cases, there would

TABLE I. ST. JOSEPH MERCY HOSPITAL ADMISSIONS FOR ACUTE CHOLECYSTITIS
(345 Patients)

Operation performed at this admission			191 Patients 9 Deaths 4.7%	
No operation performed—discharged			154 Patients No deaths	
PATIENT REFUSED OPERATION	OTHER DISEASE PARAMOUNT	NORMAL X-RAYS	MEDICAL CARE	TO RETURN FOR OPERATION
14	41	21	58	20
Returned for operation 4	7	0	24	12
47 Patients returned for operation		30%		

have been no unnecessary operation done. Such an assumption is not only unjustified but is dangerous for our hospital or any other community hospital. If immediate surgery should be adopted as a hospital policy, much ill-advised surgery would be done. In contrast, conservative treatment, done without mortality in this group, offers restraint to the surgeon and for the patient a better medical evaluation. Obviously in teaching institutions where clinical material is carefully controlled on Services, such restraint is unnecessary.

Of the 345 cases treated for acute cholecystitis, 191 were treated by operation during this hospitalization. Table II shows the type of operation performed. Surgery of election followed successful conservative treatment in 126 cases with one fatality considered due to a dissecting aneurysm. The cholecystostomy rate was 4 per cent. These figures compare favorably with Reines and Kessler,[8] and Zollinger and associates,[10] but not with Mulholland.[4] Therefore, of the 345 cases of acute cholecystitis treated conservatively, the disease was controlled in 280 (81.5 per cent), leading to elective surgery in 125 and discharge without operation in 154. There was one death.

TABLE II. ST. JOSEPH MERCY HOSPITAL EMERGENCY ADMISSIONS FOR ACUTE CHOLECYSTITIS—OPERATION PERFORMED
(191 Patients)

OPERATION	CHOLECYSTECTOMY	DEATHS	CHOLECYSTOSTOMY	DEATHS
Elective	121	1	5	0
Urgent	33	1	16	1
Emergency	5	1	11	5
Totals	159	3 (1.9%)	32	6 (18.7%)

Those patients requiring surgery not of election have been labeled the price or the risk of conservative treatment. There were 8 fatalities in these 65 patients and a cholecystostomy rate of 40 per cent. The high cholecystostomy rate is indicative of the severity of the disease. The reasons for urgent or emergency operation are shown in Table III. Those with continuing jaundice represent failure to relieve common duct obstruction by conservative means. That there were no deaths indicates good conservative management. All cases started as clinical acute cholecystitis, not as jaundice, cause to be determined. The next category, those operated upon in the judgment of the surgeon, presented the problem of a delicate balance between the cholecystic disease and the clinical condition of the patient. One death occurred from pulmonary embolism on the eleventh postoperative day as shown by autopsy. Status quo unchanged means that the control of the gall-bladder disease stopped at a safe but unsatisfactory level for discharge. The one death occurred in an 82-year-old diabetic patient on the thirtieth postoperative day, the cause undetermined except for deterioration of age. Those 27 cases with an unsatisfactory course are actually the only patients who had cholecystic disease uncontrolled by conservative treatment. There were 6 deaths; three were operated upon within 72 hours and a fourth on the fourth hospital day. The cause of death in these 6 patients as shown by autopsy

was necrotizing pancreatitis, right heart failure, and pulmonary embolism; without autopsy they were considered to be cardiac arrest with cardiac massage, pulmonary atelectasis with myocardial infarction, and possibly a "liver death" associated with deepening jaundice and a rising nonprotein nitrogen.

TABLE III. REASONS FOR URGENT OR EMERGENCY OPERATION IN 65 OF 345 CASES OF ACUTE CHOLECYSTITIS AT ST. JOSEPH MERCY HOSPITAL

CONTINUING JAUNDICE	BEST JUDGMENT OF SURGEON	STATUS QUO UNCHANGED	COURSE UNSATISFACTORY
12 cases	11 cases	15 cases	27 cases
No deaths	1 death	1 death	6 deaths

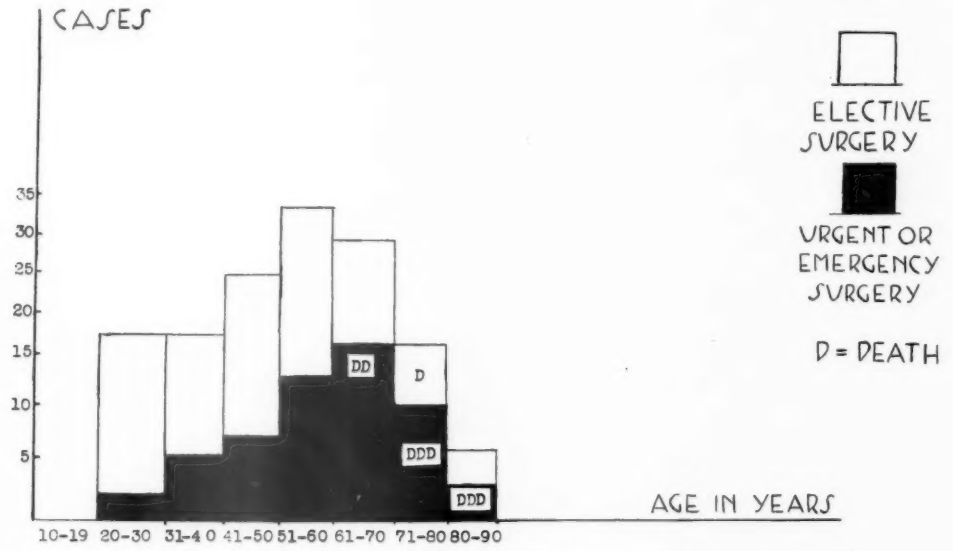

Fig. 1.—Acute cholecystitis treated conservatively and then by surgery.

Glenn,[6] in a survey of deaths following gall-bladder surgery, noted a sharp drop in those causes related to the gall-bladder surgery, that is, shock, hemorrhage, and infection, and an increase in cardiovascular and pulmonary deaths. Hepatic insufficiency remained the same. Rupture of the gall bladder and pericholecystic abscess leading to peritonitis have been used as strong arguments for immediate surgery. Glenn attributes the lack of infection as a cause of death to the policy of immediate surgery, eliminating rupture and pericholecystic abscess. With conservative treatment in this series rupture of the gall bladder is known to have occurred 4 times and pericholecystic abscess 6 times with no effect on mortality. Therefore, these possible complications do not seem valid arguments for immediate surgery. Also, we doubt that immediate operation will reduce the mortality in acute cholecystitis. Four of our deaths were in patients operated upon within 72 hours and a fifth on the fourth day of treatment. Rather as shown by Fig. 1 and emphasized by other authors, the problem is one of surgery in the aged. All deaths occurred in patients over 60 years of age and 7 of the 9 were over 70 years. The causes of death as previously shown are those hazards of any

surgery in the aged. They are grouped in Table IV. Delay by conservative treatment in this series cannot be shown to be the factor explaining the mortality. At the risk of increasing unnecessary surgery in community hospitals, the benefits of cholecystectomy in younger patients with known gall-bladder disease must be emphasized, but it is strongly doubted that immediate surgery in the aged and poor-risk patient will improve the poor results in this group of patients.

TABLE IV. DEATHS OCCURRING IN THE TREATMENT OF ACUTE CHOLECYSTITIS AT ST. JOSEPH MERCY HOSPITAL
(345 Patients)

PATIENT	AGE	OPERATION AFTER DAYS OF TREATEMNT	CAUSE OF DEATH	TIME POST-OPERATIVE	AUTOPSY
1	84	Urgent cholecystectomy at 2 days and common duct Best judgment of surgeon	Pulmonary embolism	11 days	Yes
2	70	Emergency cholecystostomy at 4 days Unsatisfactory course	Necrotizing pancreatitis	3 days	Yes
3	62	Emergency cholecystostomy at 3 days Unsatisfactory course	Right heart failure, pulmonary atelectasis	4 days	Yes
4	83	Emergency cholecystostomy at 9 days Pericholecystic abscess Unsatisfactory course	Pulmonary embolism	20 days	Yes
5	71	Emergency cholecystostomy at 1 day Pericholecystic abscess Unsatisfactory course	Cardiac arrest at operation		No
6	77	Elective cholecystectomy at 13 days	? Dissecting aneurysm	6 days	No
7	82	Urgent cholecystostomy at 12 days Status quo maintained	Undetermined other than age and slow deterioration	30 days	No
8	67	Urgent cholecystostomy at 24 hours Unsatisfactory course	Atelectasis, diabetes	8 days	No
9	72	Emergency cholecystectomy at 15 days and common duct Unsatisfactory course	? "Liver death," jaundice rising NPN coma	12 days	No

The error in diagnosis is seldom mentioned in articles advocating immediate surgery. We can add little for the error after conservative treatment due to a change in the operating room ledger of this hospital. For 2½ years of this 5-year survey we know that the preoperative diagnosis was confirmed at operation for all patients but two, who were treated conservatively for acute cholecystitis and then were submitted to surgery. In these two, at operation gallstones were not found and the pathologist reported minimal evidence of chronic cholecystitis.

During this 5-year period 8 patients operated upon as emergencies, 6 for acute appendicitis and 2 for ruptured ulcer, actually had acute cholecystitis. There was one death. The criticism that this error in diagnosis warrants is difficult to determine.

Johns,[7] Coller,[2] Boyden,[1] and others have emphasized the danger of common duct damage during surgery for acute cholecystitis. The incidence of common duct exploration is presented in Table V. The common duct was damaged once and immediately repaired. We know of no biliary cripples created after the conservative treatment of acute cholecystitis. In this series, 6 anomalies of the biliary tree were recognized and no damage was done.

TABLE V. COMMON DUCT EXPLORATION AT ST. JOSEPH MERCY HOSPITAL

OPERATION FOR CHRONIC GALL-BLADDER DISEASE		OPERATION AFTER ADMISSION FOR ACUTE CHOLECYSTITIS	
Cholecystectomy	Common duct exploration	Cholecystectomy	Common duct exploration
407	104 (25%)	159	66 (41%)
Stones recovered	16 (15%)	Stones recovered	33 (50%)

DISCUSSION

The conservative treatment for acute cholecystitis, meaning the proper use of sedation, intravenous fluids, and gastric suction instead of "watching," has no glamour and few backers. Immediate surgery, meaning operation as soon as the patient's condition will permit, is popular and advocated as a principle for surgical services. We have endeavored to show that conservative treatment as applied to 345 patients treated for acute cholecystitis has produced acceptable results that stand comparison with the results of immediate operation. Our failures that did not allow surgery of election have been described. Rupture of the gall bladder and pericholecystic abscess are known to have occurred 4 and 6 times, respectively, without contributing to the mortality. In our experience these complications are not valid arguments for immediate surgery. Our mortality as indicated by the causes of death are more closely associated with the problem of surgery in the aged than with complications of delayed surgery. Surgery was performed by 72 hours in 3 and on the fourth day in another of the 8 fatalities, indicating little delay.

Immediate surgery as a policy for services in teaching institutions can be as sound as the control of the clinical material. As a policy for the average community hospital where the majority of gall-bladder surgery of this country is done, it could lead to much ill-advised surgery. In community hospitals, certain individuals of outstanding ability can certainly apply the principle of immediate surgery to their own practice with success but they must also take the responsibility for their imitators.

SUMMARY

1. The results of the conservative treatment of 345 patients with acute cholecystitis have been presented as a defense of this treatment.

2. These 345 patients were treated in a 5-year period, 1952 through 1956, at St. Joseph Mercy Hospital, Ann Arbor. Those patients with adequate history, physical findings, and laboratory work to warrant treatment for acute cholecystitis were considered to have had acute cholecystitis and make up this series. Not included are those who were erroneously so diagnosed and later were proved to have had another disease. Not included are those who were treated for another disease and later were shown to have had acute cholecystitis.

3. Of these 345 cases the disease was controlled in 280 (81.5 per cent), leading to elective surgery in 125 and discharge without surgery in 154. There was one death. Thirty per cent of those discharged without surgery returned for surgery.

4. Because conservative treatment did not bring satisfactory remission, urgent or emergency surgery was required in 65 cases. There were 8 deaths. The reasons for urgent or emergency surgery are listed.

5. A review of the deaths indicates that surgery in the aged is the problem rather than complications of delayed surgery in gall-bladder disease.

6. Immediate operation as a policy for a Service in a teaching institution is undoubtedly satisfactory. The dangers of such a policy for the average community hospital are emphasized and the conservative treatment proposed as a restraint to the average surgeon and, moreover, for the patient a better medical evaluation. A policy of conservative treatment for acute cholecystitis will give acceptable results.

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A SIMPLE TECHNIQUE FOR PREPARING VAGALLY INNERVATED GASTRIC POUCHES IN DOGS

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ALTHOUGH dogs with gastric pouches are extensively utilized in investigation of gastric secretory function, most workers have chosen vagotomized (Heidenhain) stomach pouches for study rather than preparations with the vagal innervation preserved (Pavlov).[6] This choice has been dictated, in part at least, by the difficulties involved in preparing satisfactory Pavlov pouches. In the usual techniques for making a Pavlov pouch,[1, 2, 4-7] a seromuscular bridge is preserved between main stomach and pouch and separation is achieved by closing the mucous membrane in two layers. Fistula formation between stomach and pouch occurs rather easily, resulting in a useless preparation. Also, such pouches cannot be utilized very satisfactory for experiments involving surgical procedures on the stomach.

Recent investigations of available methods for construction of Pavlov pouches in dogs led to development of the technique to be described, which is a simple yet satisfactory method for obtaining vagally innervated gastric pouches. Animals so prepared can be operated upon subsequently in the same manner as animals with Heidenhain pouches.

DESCRIPTION OF METHOD

Large dogs are utilized. The animal is anesthetized with sodium pentobarbital (30 mg. per kilogram) and intermittent positive pressure respiration is established through an endotracheal tube. With aseptic precautions, the left chest is opened through the tenth intercostal space and the abdomen entered through the left leaf of the diaphragm. The esophageal hiatus is opened and the anterior vagus trunk is then carefully dissected away from the lower esophagus and followed distally to its initial branching to the anterior wall of the fundus of the stomach. These branches are preserved, dissecting them up only enough to provide an area for subsequent transection of the stomach (Fig. 1, *A* and *B*). The distal portion of the anterior vagus trunk directed toward the lesser curvature is then sectioned. The uppermost branches from splenic and left gastroepiploic arteries are carefully preserved as these vessels will subsequently supply the pouch. The stomach wall at the site of proposed division is prepared

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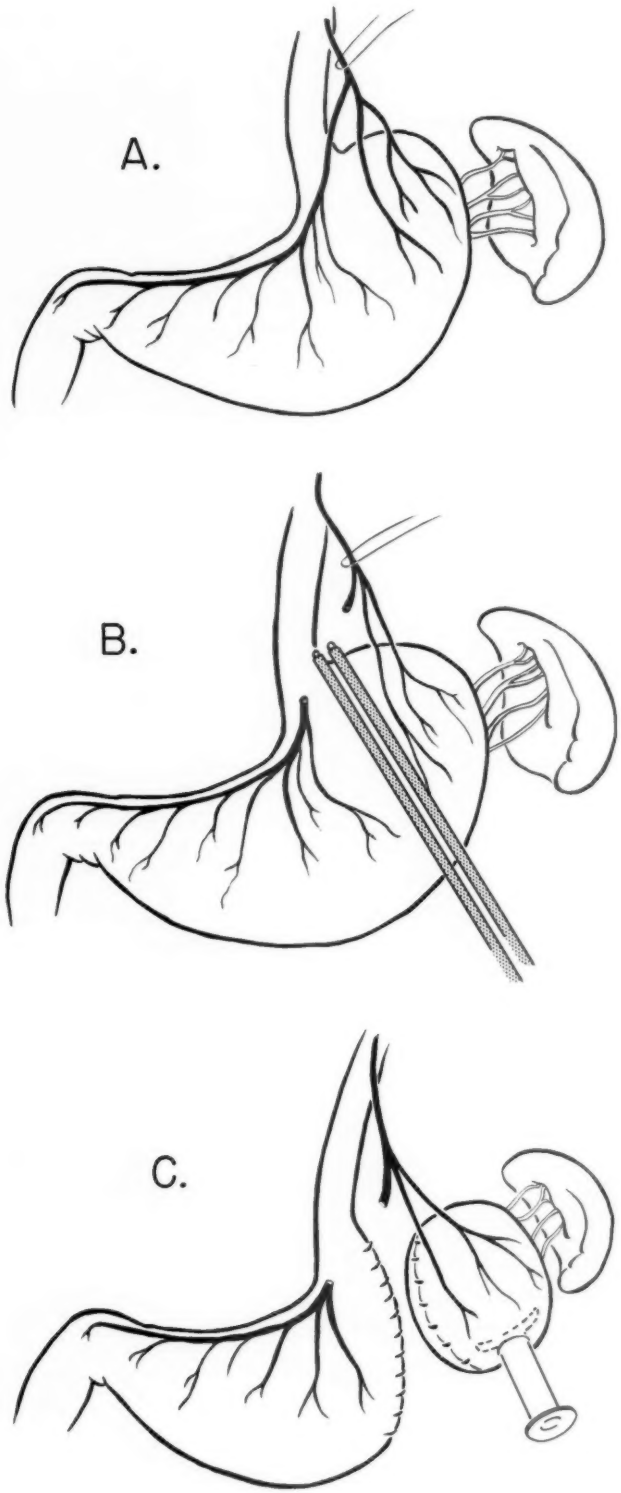

Fig. 1.

and is transected between clamps with thin blades (Fig. 1, *B*). A greater portion of anterior than of posterior stomach wall is included on the pouch side of the clamps. This is easily accomplished by so positioning the stomach wall with the fingers when the clamps are applied. This maneuver is important as it results in a pouch with proportionately greater innervation of the wall since no effort is made to preserve the integrity of the fundic branches of the dorsal vagal trunk. By oversewing the cut edge of both pouch and residual stomach before removing the clamps, significant bleeding is prevented. Lembert sutures of fine silk complete the closures (Fig. 1, *C*). During closure of pouch and stomach the vagal branches are carefully protected. A plastic cannula is placed in the pouch through a separate opening and brought out through a stab wound in the anterolateral abdominal wall. The pouch is fixed to the abdominal wall at this point with a few sutures to prevent torsion or tension on the vagal fibers or vessels. The incisions are closed in the conventional manner and the usual postoperative care for animals undergoing gastric operations is carried out. Following operation, the animals are allowed a period of convalescence of at least 3 weeks before study of pouch secretions is begun. Representative responses of pouches prepared in this manner are shown in Table I. Fasting secretion during a 2-hour period is compared with that obtained for a similar time period after insulin administration.

TABLE I. RESPONSES OF VAGALLY INNERVATED GASTRIC POUCHES FOLLOWING INSULIN ADMINISTRATION

	2-HOUR FASTING SECRETION		2-HOUR SECRETION FOLLOWING INSULIN*	
ANIMAL	VOLUME (ML.)	FREE HCl (MEQ.)	VOLUME (ML.)	FREE HCl (MEQ.)
1	3.0	0	13.0	0.97
2	2.0	0	1.6	0.08
3	0.4	0	8.0	0.32
4	6.0	0	20.0	1.4
5	3.0	0	9.0	0.63
6	4.0	0	8.0	0.40

*Ten units crystalline insulin given subcutaneously 15 minutes prior to 2-hour collection period.

DISCUSSION

The method described depends upon careful nontraumatic dissection of the anterior vagal trunk and its branches. Obviously, damage to this structure will result in a functionally denervated preparation. However, it has not proved especially difficult to isolate and preserve this nerve. The majority of animals so prepared have shown positive responses to insulin administration. As the dorsal vagus trunk is not disturbed, the stomach retains at least half of its normal vagal innervation. Since stomach and pouch are individually closed in two layers, internal fistula does not occur. This would appear to be the principal advantage of this method over several previously described technical modifications of Pavlov's pouch. In addition, animals can be used for experiments involving additional surgery without disturbing the pouch. Pouches prepared in this manner are about the size of conventional Heidenhain pouches.

SUMMARY

A technique for preparation of vagally innervated gastric pouches is described wherein the branches of the anterior vagus trunk to the fundus are preserved and the fundic pouch is separated completely from the stomach. Such pouches can be shown to respond in a positive manner to the insulin test.

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TWO CASES OF POSTERIOR MEDIASTINAL GOITER AND THEIR MANAGEMENT

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POSTERIORLY situated goiter in the superior mediastinum is unusual enough to merit special consideration as an entity distinct from the more commonplace substernal thyroid enlargement. The embryologic origin of the condition which we report is not clearly established. It may result from failure of complete fusion and ascent of the lateral ultimobranchial bodies with the main primordium of the thyroid gland in the embryo. Alternatively a goiter so located may be deflected posteriorly by the great vessels and transverse arch of the aorta as the thyroid enlargement "plunges" into the thorax. These hypotheses are considered by Sweet in his report of 6 such cases.[3] We have found an additional case report by Crohn and Kobak[1] as well as 2 further cases reported by Hurwitz and Skorneck.[2]

A close study of the radiograms and anatomic sketch derived from our first patient draws attention to relationships which are consistently present in all reported cases. These facts should permit accurate preoperative diagnosis. The thyroid mass displaces the esophagus posteriorly and the trachea anteriorly so that a "common wall" no longer exists between these two organs. The goiter is behind the great vessels to the upper extremities and head, and to the right of the transverse arch of the aorta. The arch of the azygos vein will be seen stretched over the lateral aspect of the tumor as it presents in the right hemithorax. The lowermost extent of the tumor may reach as far as the inferior pulmonary vein. These complicated and hazardous anatomic relationships make any approach other than a transthoracic one to the lesion inconceivable.

Because of the wrist-sized connection between the tumor and the lower pole of the left lobe of the thyroid in our first case, the successful removal of the tumor required both a separate right thoracic and a cervical incision.

CASE REPORTS

CASE 1.—L. R., an obese, 40-year-old, white, married man, had noted shortness of breath for 2½ years. This could be relieved by lying on his left side or leaning forward from a sitting position. There had never been difficulty in swallowing or hemoptysis. For 7 days his cough had been much worse and he had been unable to lie flat because of a sense of asphyxia. Family history, past history, and systemic review were unremarkable. Physical examination revealed an anxious, obese man in moderately severe respiratory distress. Pulse was 80; blood pressure was 170/110. Respirations were 24 per minute, were punctuated by cough, and were associated with an audible wheeze. The trachea was palpably shifted to the right. There was no retrosternal dullness to percussion or suprasternal mass to palpation.

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Auscultation of the lungs revealed a persistent wheeze and coarsened breath sounds of stertorous quality without crepitant râles. The remainder of the physical examination was normal. The laboratory work was not abnormal. X-ray examination of the chest demonstrated a large mass in the posterior mediastinum extending from the thoracic inlet to a level below the carina. This mass displaced the trachea anteriorly and to the right and the esophagus posteriorly and to the right (Fig. 1, *A* and *B*). On Aug. 30, 1956, a right posterolateral thoracotomy was performed through the bed of the fifth rib. A large, roughly elliptical, pale, grayish-red mass was encountered extending into the posterior mediastinum from the neck to a level just cephalad of the inferior pulmonary vein. This measured 7 by 10 by 4 cm. in greatest extent. It displaced the esophagus laterally into the right chest and the trachea anteriorly. A much lengthened arch of the azygos vein was stretched out over the lateral aspect of the tumor. There was no major vascular supply to the tumor within the chest. A 4 cm. "waist" of the tumor extended to the left behind the innominate vessels and aortic arch into the base of the left side of the neck (Fig. 2). After the tumor had been freed of its attachments, its waist was amputated at the point where it entered the

A. *B.*

Fig. 1.—Posteroanterior (*A*) and lateral (*B*) chest x-rays showing mediastinal goiter displacing esophagus and trachea.

neck. Bleeding from this very vascular cut surface was controlled by mattress sutures. The chest was closed. The patient was turned from the left lateral decubitus to the supine position. A classical, low, cervical collar-type incision was made and a left hemithyroidectomy was performed. The 4 cm. waist attached to the lower pole of the left lobe of the thyroid easily was brought into the neck. The blood supply of the lesion was exclusively derived from the left inferior thyroid artery. Gross pathologic examination showed an encapsulated 10 by 7 by 4 cm. mass consisting of very gelatinous material and weighing 385 grams. Microscopic examination showed a colloid goiter. The interesting anatomic situation of the lesion in situ is clearly shown in the composite sketch (Fig. 3).

CASE 2.—M. H., a 58-year-old white, married woman, had an annual check-up by her physician one week prior to her admission. A chest x-ray disclosed a mediastinal mass extending from the base of the neck into the chest deviating the trachea anteriorly and the

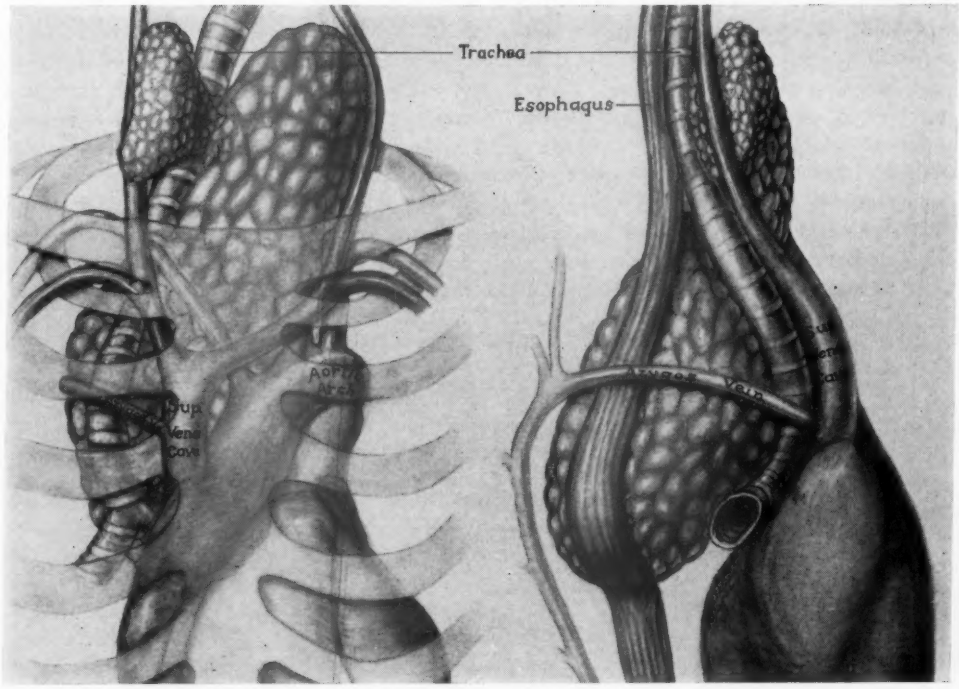
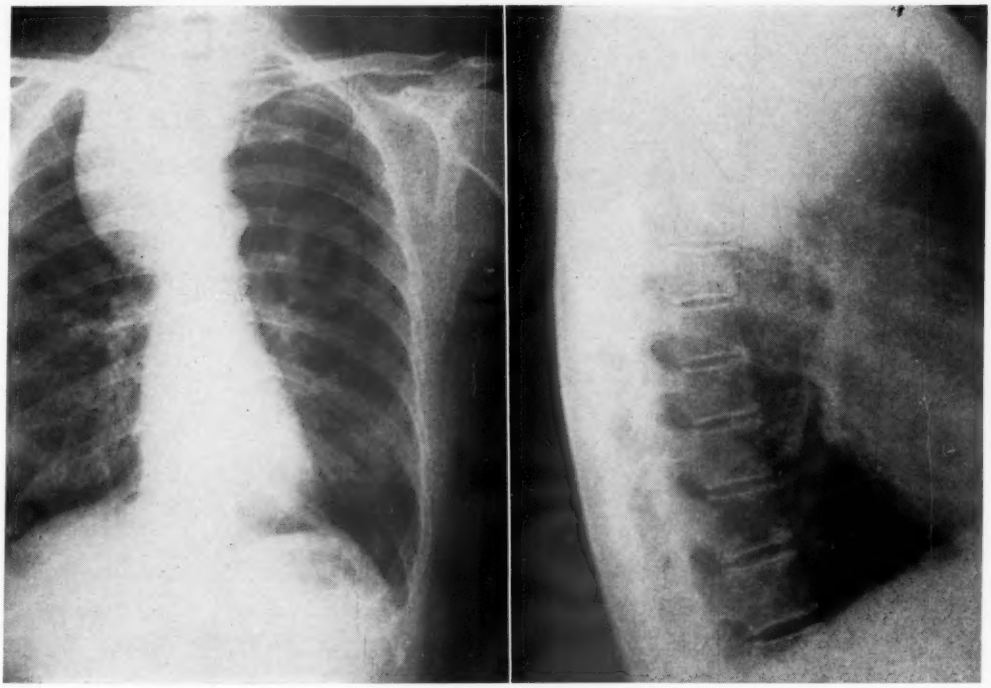


Fig. 2.—Artist's sketch of anatomic relationships of this goiter.



A. *B.*

Fig. 3.—Posteroanterior (*A*) and lateral (*B*) chest x-rays showing posterior mediastinal goiter. No tracheal displacement.

esophagus posteriorly and to the left. Faint calcification was seen within the mass (Fig. 3). The patient had been completely asymptomatic before this accidental discovery. Physical examination was normal. Routine laboratory studies were normal. A standard posterolateral thoracotomy was performed. An 11 cm. mass was encountered coming from the inferior pole of the right lobe of the thyroid, through the neck, into the chest, and behind the innominate vessels and aortic arch. The trachea was deviated anteriorly and the esophagus posteriorly and to the left. The tumor reached inferiorly to the arch of the azygos vein. It was removed without difficulty because it was well encapsulated and the stalk attaching it to the right lobe of the thyroid was narrow. The gross pathologic examination showed an encapsulated, reddish, gelatinous mass with focal calcified elements weighing 215 grams. The microscopic examination revealed colloid goiter.

SUMMARY

Posterior mediastinal goiter, though unusual, presents with sufficiently characteristic roentgen findings to make the preoperative diagnosis possible. This type of goiter because of its hazardous and complicated anatomic relationships should be removed through a chest incision. At times both thoracic and low cervical incision may be required. Illustrations depicting the salient features of the lesion are presented.

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PRIMARY FISTULOTOMY IN THE TREATMENT OF ANORECTAL ABSCESSES

Report of One Hundred Consecutive Cases

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FOR many years there has been difference of opinion among surgeons and some proctologists concerning the treatment of anorectal abscesses. One school of thought teaches simple incision and drainage of an abscess followed by a period of waiting before fistulectomy. The other school suggests primary fistulotomy if the abscess is simple or if the tract is superficial.

It is the purpose of this paper to report a series of 100 consecutive anorectal abscesses treated in each instance by primary fistulotomy in an effort to achieve a base line for further studies.

Table I. Location of Abscess

Perianal	51
Postanal	30
Ischioanal	8
Horseshoe	2
Intersphincteric	4
Submucous	3
Supralevator	2
Retrorectal	2
Total	100

There were 81 male and 19 female patients in this series. They were all white with an average age of 38 years. The average duration of symptoms was 10 days. Table I shows the location of each abscess. Superficial postanal space abscesses were included in the perianal group, as their treatment was the same.

Table II. Previous Operations

	NUMBER	PER CENT
Abscess	12	12
Fistulectomy	4	4
Hemorrhoidectomy	8	8
Total	24	24

Twenty-four of these patients, or 24 per cent, had undergone previous operations as can be seen in Table II. None of the surgical procedures had been performed within 6 months of the appearance of the abscess. The primary or internal openings were posterior in 58, anterior in 37, and lateral in 5.

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Of the 100 cases, 70 required division of a portion of the rectal sphincter. Between one-sixth and two-thirds of the sphincter muscle was divided in these cases. Six patients required a second procedure for removal of a seton tie. A seton tie was utilized in 6 cases when more than half of the sphincter was traversed by the fistulous tract. In recent cases primary fistulotomy without a seton tie has been performed through three-fourths of the anal musculature without ill effect.

Bridging and pocketing which occurred in 3 patients was easily corrected in the office postoperatively. Three of the patients, however, required additional anorectal surgery. One developed a recurrent abscess. This particular patient had a large retrorectal abscess which was drained posteriorly and externally. He developed purulent material in each ischioanal fossa with spread to one supralevator space. Bilateral drainage sites were established over each ischioanal space. The wounds healed promptly with the exception of a small defect deep in the posterior anal canal which has persisted for many months. This is gradually healing, however, and the patient is asymptomatic. He states that he feels well and exhibits no incontinence despite the severence of two-thirds of the anal musculature. There were 2 additional patients who developed higher internal openings following the primary procedure. These were both posterior and evidently were primary openings not demonstrated at the original procedure. Both of these healed and have remained asymptomatic.

Thus, of the 100 patients upon whom primary fistulotomy was performed, together with incision and drainage of the rectal abscess, only 3 required additional operative treatment. The length of follow-up care to date has averaged 14 months.

TABLE III. COMPLICATIONS FOLLOWING FISTULOTOMY WITHOUT ABSCESS

	NUMBER	PER CENT	THERAPY	RESULT
Bridging	3	3	Office	Good
Recurrent fistula	2	2	Operation	Good in one Poor in one
Persistent fissure	3	3	Office	Good
Abscess	0	0		
Total	8	8		

Table III shows the complications in 100 consecutive patients who underwent fistulotomy without an abscess.

Here it may be noted that the over-all complication rate was 8 per cent as against 6 per cent for the seton-tied series. Two patients were reoperated upon for recurrent fistulas. One operation was for unrecognized ileitis and the other for a missed tract, the latter patient having undergone three previous procedures elsewhere.

In 61 of the patients who underwent primary fistulotomy in the presence of abscess formation, additional anorectal surgery was performed: hemorrhoidectomy in 57 and cryptectomy in 4. None of these patients evidenced any increased morbidity.

The result was good in 97 patients and fair in 3. One fair result occurred in the patient who was operated upon for a recurrent abscess and exhibited slow, asymptomatic healing. The other 2 fair results were obtained in female patients who evidenced chronic perianal abscesses located anteriorly. Two-thirds of the musculature was incised in one case and one-half in another. Although each patient is continent of feces and possesses good voluntary control, there is occasional leakage and difficulty in controlling flatus. Sphincteroplasty will be performed in both cases shortly if better function is not forthcoming.

COMMENT

Although it is obvious that no final conclusions can be drawn from the clinical results obtained in this relatively small series, several facts of significance may be noted. Each of these patients appeared to tolerate the combined procedure very well. They were all relieved of pain and often did not manifest the moderate discomfort which follows elective fistulectomy or hemorrhoidectomy. Locating the primary or internal opening was not as difficult as anticipated. In most instances this opening, which usually was a crypt, was demonstrated from the inside by application of a bent probe into the suspected area. Dimpling, scarring, and irregularity suggested the correct location of the opening. Rarely was the opening demonstrated through the abscess cavity. The postoperative course did not vary from that of other patients undergoing anorectal procedures by the author. There was no increase in morbidity and no evidence of toxemia at any time although antibiotics were not given in any case. One patient, who was operated upon in an early acute stage for a perianal abscess, developed a small pocket of pus under the edge of the wound postoperatively. This healed promptly and may have been due to overzealous probing.

There has been some mention in the literature of excessive muscle retraction from primary fistulotomy. It has been claimed that the sphincter may retract excessively when incised in the presence of inflammation, and may retract to a lesser degree during an elective fistulotomy. I was not able to substantiate this fact and found that healing appeared to take place quite rapidly with very little retraction of muscle fibers following primary fistulotomy. This was particularly true in acute abscesses where no thickened tract had developed.

In long-standing or chronic abscesses, care should be taken to excise the walls of the tract from the underlying muscle to allow better coaptation. Although the argument has been advanced, that if the posterior wall of the tract extending through the muscle is left intact, the muscle fibers will not retract, I feel that the opposite is true and that coaptation is prevented by allowing this tissue to remain. The scar formed from the posterior wall of the tract may expand in time and cause delayed incontinence. Muscle retraction would appear to be discouraged also by removing all packing within 24 hours. Although

incision of muscle fibers was attempted always at right angles in this series, diagonal sphincterotomy was employed frequently without the development of incontinence.

Two retrorectal and one supralevator abscesses were drained internally with wide saucerization of rectal mucosa and extension of the incision to the perianal skin with partial sphincterotomy and primary internal fistulotomy. Adequate drainage and prompt healing ensued in each instance.

SUMMARY

1. An abscess about the rectum should be treated an an abscess elsewhere in the body. Immediate incision and drainage are required before necrosis of overlying tissue adds the chance of additional toxemia.

2. Primary fistulotomy appears safe if the abscess is drained correctly and saucerized widely.

3. As much as two-thirds of the sphincter mechanism may be severed posteriorly in the presence of acute inflammation. Care should be taken not to sever more than one-third of the sphincter anteriorly.

4. In small acute abscesses where a minimal amount of pus is present and walling-off may be incomplete, manipulation and probing should be minimal.

5. Concomitant anorectal surgery such as hemorrhoidectomy appears to offer no increase in morbidity but in general should be reserved for removal of obvious pathologic structures or structures which might impede drainage.

In conclusion, there has been no complication of any nature in the last 75 of these 100 operations, subsequent to the development of better technique. It is obvious and mandatory that a procedure like this should not be performed by anyone unfamiliar with anorectal anatomy and postoperative care. Additional follow-up will be continued and reported at a later date.

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MANAGEMENT OF THE PATIENT DURING OPEN HEART SURGERY

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THE continued use of the pump-oxygenator to effect bypass of the heart and lungs has widened the scope of cardiac surgery. Early clinical reports were concerned with the correction of congenital heart lesions, primarily in children.[7, 15, 18, 19] Because of increased perfusion capacity of the pump-oxygenator and improved techniques, the more complicated congenital lesions and the acquired valvular lesions can now be corrected by means of direct vision surgery. This report presents our experiences with the first 200 open heart operations, with special emphasis on the newer aspects of the management of anesthesia.

The Extracorporeal Apparatus.—The Kay-Cross pump-oxygenator† (Fig. 1) was used in all cases in this series. It consists of a rotating disc-type oxygenator[13] and suitable arterial and venous pumps. Modifications of the original extracorporeal apparatus have been made to provide more efficient oxygenation and perfusion.

In about one-half of the cases Sigmamotor pumps were employed, but more recently De Bakey-type roller pumps have been used. Although clinically the Sigmamotor pumps proved satisfactory, laboratory studies[11] of pump pressure curves, pump output, and hemolysis have led us to the clinical use of the two-roller pump. A length of siliconized latex tubing is placed in each pump with extensions of 5 to 10 inches to act as pulse dampeners. The inside diameter of the tubing is varied, depending upon the expected perfusion rate of the individual case. The pumps are adjusted to be "just" nonocclusive.

The intracardiac field is aspirated with an accessory coronary sump pump which returns the blood to the oxygenator via a defoaming chamber. Two sump pumps are used for mitral valve surgery. A filter is placed in one of the sump lines to prevent aspiration of calcium, vegetations, or thrombi into the oxygenator.

A major modification of the oxygenator has been the use of convoluted discs[14] (Fig. 2). These discs add about 40 per cent greater gas exchange surface at the same disc rotation rate. This increases the oxygenating ability by approximately 30 per cent without increasing priming volume or hemolysis.

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†Manufactured by Pemco Company, Cleveland, Ohio.

Extracorporeal Perfusion.—The management of the patient during open heart surgery, with the use of a pump-oxygenator, requires a high degree of teamwork, understanding, and liaison between various members of the cardiac surgery team. It is important to have some one individual responsible for coordinating the various components of the team, whether he be a surgeon, cardiologist, physiologist, or anesthesiologist. In our opinion, it is advantageous for the surgeon to be able to concentrate his efforts on the operation itself. The anesthesiologist is in the best position to observe the clinical condition of the patient and the effects of total body perfusion, by direct observation of the patient, as well as observation of the various monitoring devices.

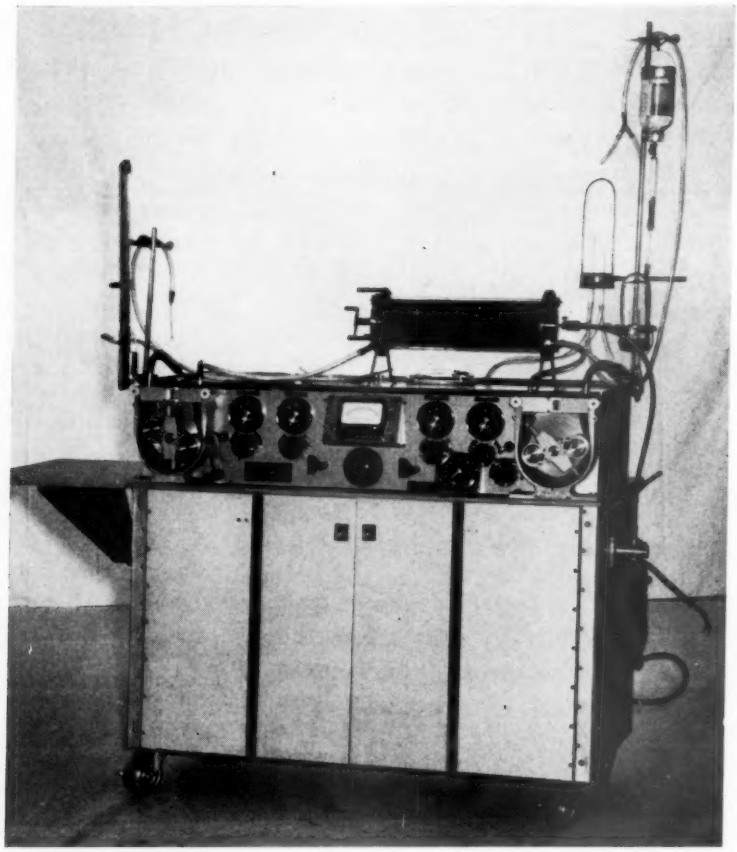

Fig. 1.—Present model of the Kay-Cross oxygenator.

Anesthesia.—The anesthetic has been primarily endotracheal cyclopropane in a closed circle filter employing controlled hyperventilation. The details and rationale of this anesthesia have been previously described.[21] During bypass, anesthesia is maintained by the continous addition of cyclopropane, along with carbon dioxide and oxygen to the oxygenator. Cyclopropane blood levels were determined by the method previously described by Orcutt and Waters[23] from arterial samples drawn before, during, and following bypass. The concentration of cyclopropane in the arterial blood averaged 6.5 mg. per cent (range 4 to 8

mg. per cent). This is consistent with electroencephalographic levels 1 or 3 usually observed and the clinical evaluation of the patient. This permits a quiet field, no movement of the diaphragm, with a minimal amount of anesthesia. On several occasions, patients have remembered events which occurred during bypass; they report an awareness of time and place, but a lack of pain and fear.

In different cases, during bypass, the lungs have been partially inflated with minimal intermittent positive pressure, ventilated with oxygen, or allowed to collapse completely. There has been no evidence that pulmonary complications have been produced or aggravated by any of these methods. However, in patients with large collateral bronchial flow, for example, tetralogy of Fallot, positive pressure on the airway is avoided as it will often flood the operative field with pulmonary reflux blood.

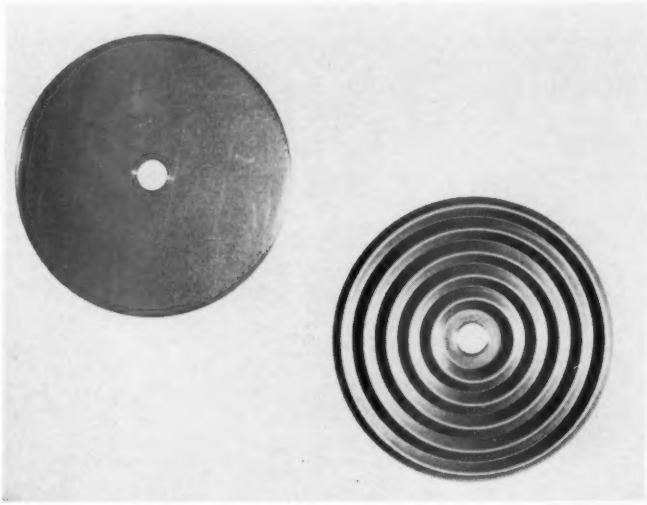

Fig. 2.—Stainless steel discs. Left, Flat disc. Right, Convoluted disc which increases oxygenation surface area 40 per cent.

Supplemental Drugs.—A number of supplemental drugs have been used when complications have occurred during surgery. All drugs are given intravenously except during bypass, at which time they are added to the arterial end of the oxygenator. Aramine (metaraminol bitartrate) has been the most useful vasopressor. Before and after the bypass period, Aramine (1 mg.) is used in the treatment of hypotension. This is in conjunction with correction of the cause of the hypotension. During the bypass period, Aramine has frequently been effective in increasing perfusion pressure.

Vasoxyl (methoxamine hydrochloride) is avoided except during the bypass period because of its lack of inotropic effect on the heart.[3] Norepinephrine is used infrequently and has been of little use in the series. As previously reported, Adrenalin and calcium chloride have been useful cardiac stimulants.[21]

Isuprel (isoproterenol) is effective in the treatment of centricular fibrillation which follows elective cardiac arrest. With continued perfusion effective

systole followed injection of Isuprel into the oxygenator in 7 cases. In 2 of these cases, chemical defibrillation with Isuprel was successful after repeated attempts at defibrillation with electric shock were unsuccessful (Fig 3).

Succinylcholine chloride is the only relaxant drug used. It is used (1) to facilitate intubation of adults, (2) to prevent massive muscle contractions during electric defibrillation, and (3) occasionally to control persistent diaphragmatic movement during bypass.

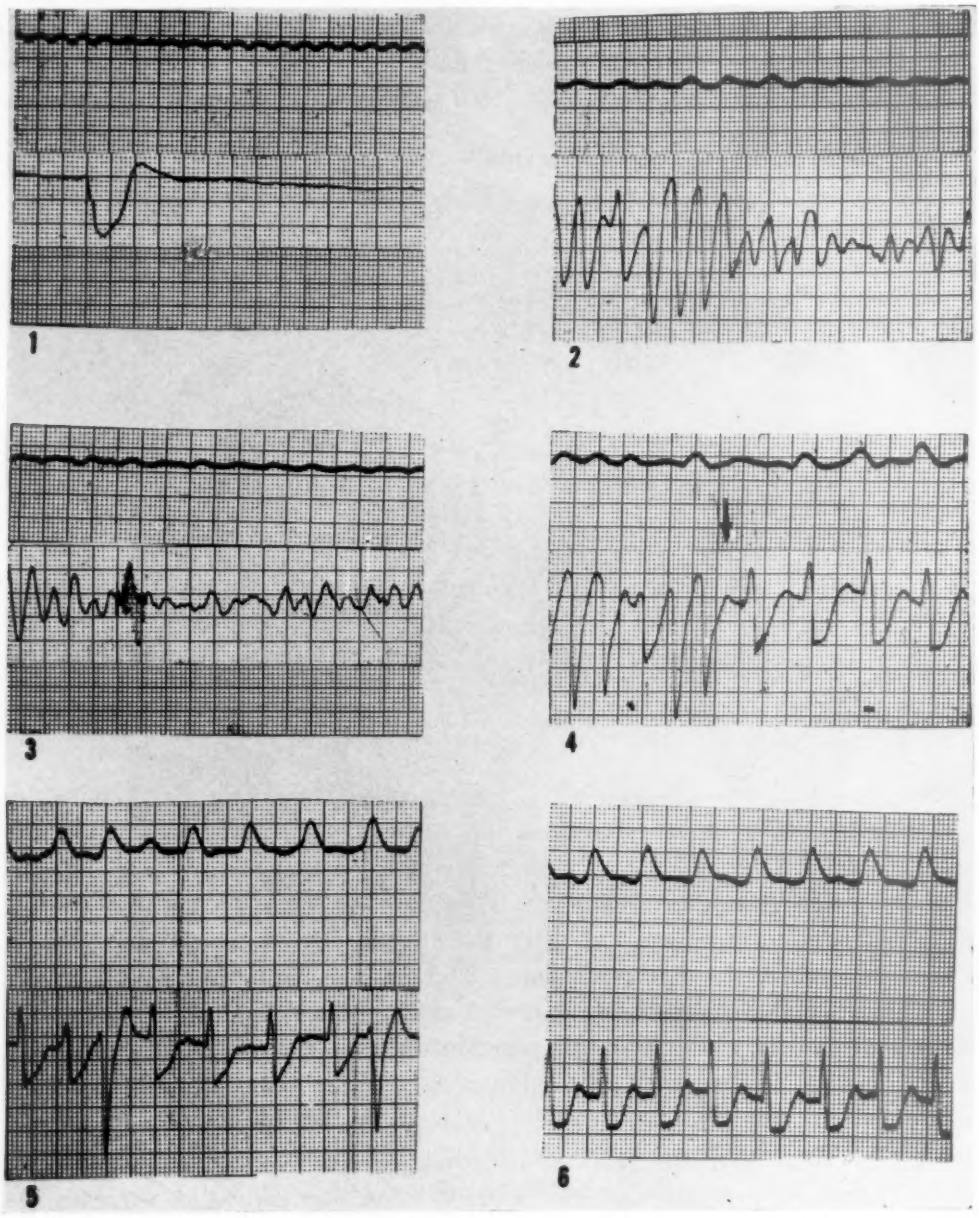

Fig. 3.—Electrocardiogram (Lead II) and arterial blood pressure of 32-year-old male during total bypass for the correction of rheumatic aortic stenosis. *1*, Potassium arrest. *2*, Ventricular fibrillation following removal of aortic clamps after 19 minutes. *3*, Ventricular fibrillation with repeated attempts at electric defibrillation. *4*, Defibrillation occurring 30 seconds after Isuprel (0.1 mg.) added to the oxygenator. *5* and *6*, Recovery period.

Physiologic Measurements.—A number of monitoring aids have been employed to observe physiologic changes and aid in their management, especially during bypass (Fig. 4). Electrocardiograms and electroencephalograms are viewed continuously during operation on an oscillograph screen.* These are recorded at intervals, or during unusual occurrences, on a Sanborn 150 recorder. The electrocardiographic findings have been previously reported.[27] Arterial pressure is measured and recorded with a Statham transducer, Model P23AA, and the Sandborn recorder. Superior vena caval pressures are measured with a water canometer. In small infants the introduction of a superior vena caval catheter, particularly in the presence of a midline incision, may be more difficult, so consequently an inferior vena caval catheter is introduced into the inferior vena cava through the superficial femoral vein at the time of the femoral cannulization. Inferior vena caval pressures may be higher and more affected by the depth of anesthesia and abdominal wall tension than superior vena caval pressures. The correlation between superior and inferior vena caval pressures and the factors influencing them during the extracorporeal perfusion are the subject of a separate report.

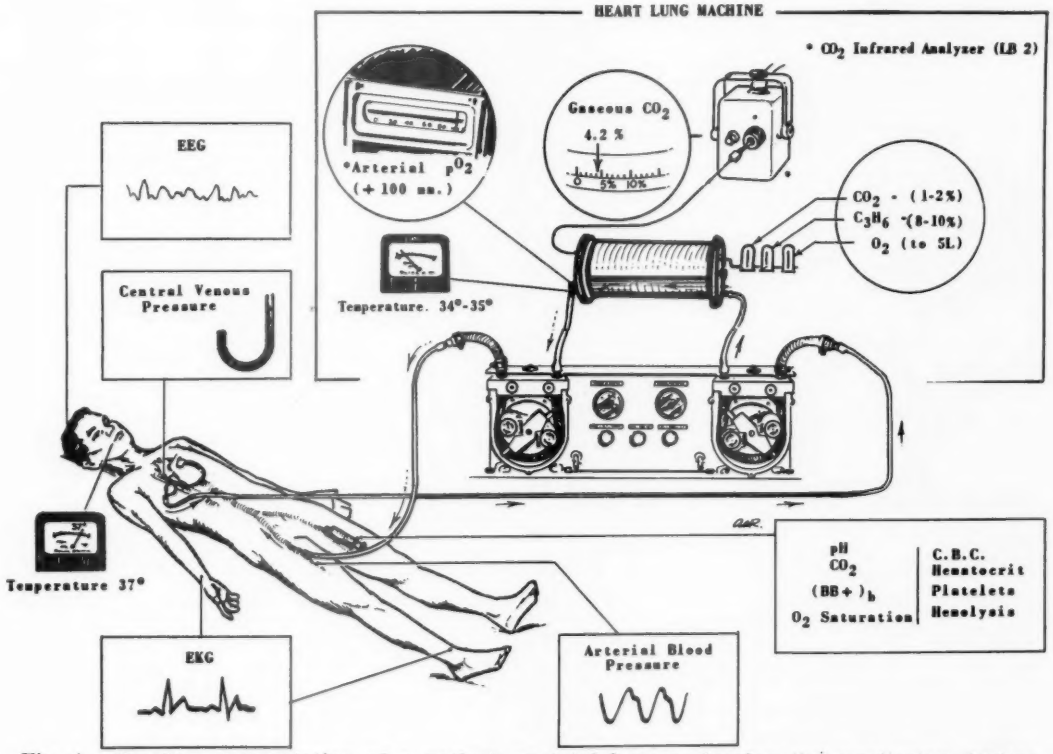

Fig. 4.—Graphic representation of a patient on total bypass showing the monitoring devices and arterial blood determinations.

Esophageal and oxygenator blood temperatures are monitored with a Yellow Springs thermistor. During the research phase, as well as early in the clinical use of extracorporeal circulation, special emphasis was placed on keeping the

*Monitorscope by Essential Electronics, Cleveland, Ohio.

oxygenator and body as close to normal temperatures as possible. This was occasionally difficult in small infants in an air-conditioned room even when the oxygenator was heated and the infant placed on a heated mattress. Less stress has been placed upon this aspect recently, the main consideration being equalization of temperature in the oxygenator and body, so as to avoid the theoretical consideration of oxygen bubbles in instances of high oxygen tension when the body might be warmer than the oxygenator.

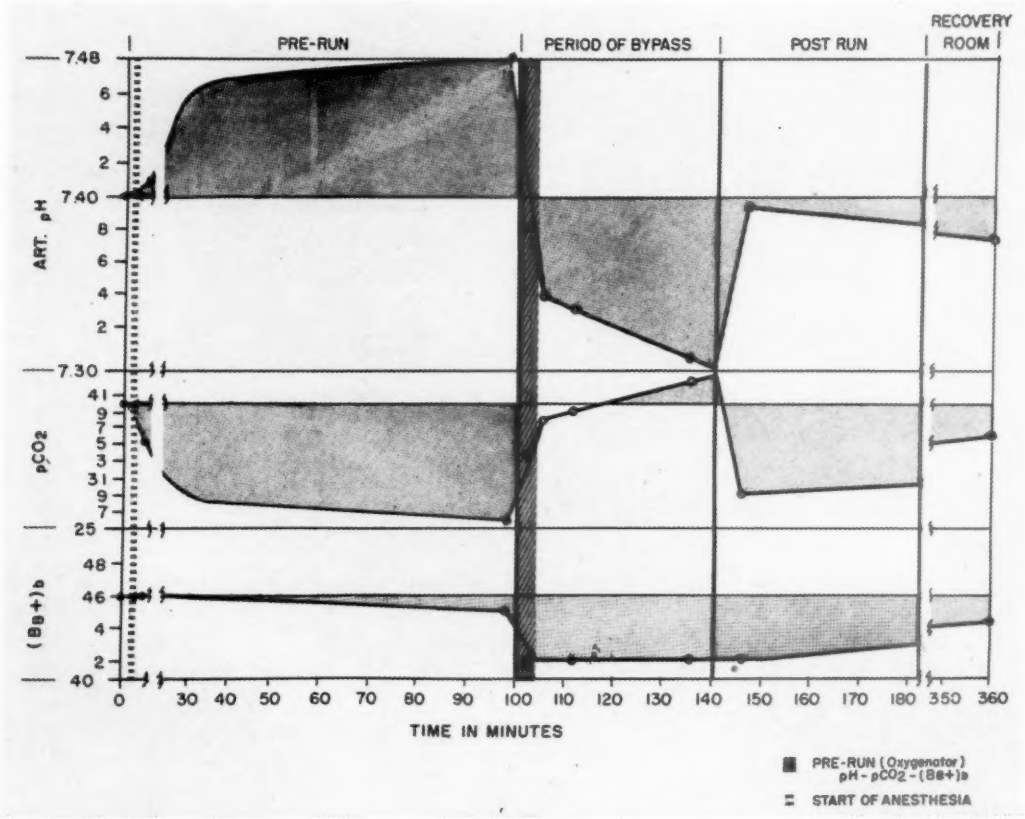

Fig. 5.—Graph representing average values of arterial pH, pCO_2, and blood buffer cases in Cases 100-150.

Oxygen tension of the arterial blood leaving the oxygenator is measured by a Clark oxygen polarograph.[6] Carbon dioxide concentration of the effluent gases in the arterial end of the oxygenator is measured and recorded with a Beckman CO_2 infrared analyzer, Model LB 2. The amount of carbon dioxide added to the oxygenator is sufficient to result in a CO_2 concentration in the effluent gases of 3.8 to 4.2 per cent. This in turn gives an arterial carbon dioxide tension of approximately 40 mm. Hg at the temperature at which the oxygenator is operated (33 to 35° C.). In addition to the above continuous measurements, intermittent arterial samples are drawn for determination of pH, oxygen, and carbon dioxide content, before, during, and following surgery (Fig. 5).

Series.—This report is based upon the first 200 heart operations which have been performed on 190 patients, utilizing the pump-oxygenator (Table I). Six patients of this series were operated upon twice. Two patients were operated upon three times. In 7 of these patients, the defect had reopened or had been incompletely corrected. One patient was operated upon twice to resuture a ruptured mycotic aneurysm of the right ventricle due to a Pseudomonas myocarditis which developed following closure of a ventricular septal defect.

TABLE I. ANATOMIC DIAGNOSIS, 200 CASES

CONGENITAL LESIONS		ACQUIRED LESIONS	
LESION	NO. OF CASES	LESION	NO. OF CASES
IVSD	36	Mitral insufficiency (predominant)	18
IASD	27	Mitral stenosis (predominant)	14
IVSD and pulmonic stenosis	28	Myoma of atrium	1
IVSD, reoperation, tetralogy	5	Mitral and tricuspid disease	3
Pentalogy	4	Aortic and mitral disease	8
A-V communis and mitral insufficiency	6	Aortic insufficiency	3
Pulmonic stenosis	15	Aortic stenosis	1
Transposition of great vessels	4	Lutembacher syndrome	1
IVSD and IASD	2	Miscellaneous	2
Aortic stenosis	3		
Miscellaneous	19		
Total	149		51
Total mortality (all cases) 28%			

The patients' ages ranged from 1 month to 54 years. The patients' weights ranged from 3 to 90 kilograms. One hundred forty-nine operations were performed for the correction of congenital heart lesions; 51 for the correction of acquired lesions.

There is an over-all mortality rate of 28 per cent ranging from no mortality in noncomplicated congenital defects to 100 per cent in the more complicated defects such as transposition of the great vessels. Thus, the mortality rate is primarily influenced by the type of lesion to be corrected.

Flow Rates.—During the past there have been those who have advocated low flow rates and those who have advocated high flow rates. The oxygen requirements of the body are not satisfied when low flow rates are used and in instances of prolonged perfusion this undoubtedly leads to metabolic acidosis. On the other hand, nothing is gained from using flow rates greater than necessary to fulfill basic requirements and may even be detrimental as far as blood destruction and bleeding tendencies are concerned. As a consequence, we have always followed an intermediate policy with flow rates, dependent upon body size, oxygen tension, arteriovenous difference, and mean arterial pressure.

Blood Volume Control.—It is axiomatic that successful extracorporeal perfusion is dependent upon the maintenance of the patient's normal blood volume. Unless this is closely observed and maintained, an increase or decrease in circulating blood volume may not only make it difficult or impossible to control the patient physiologically during bypass, but may be an important factor

in the heart's ability to take over afterward. Blood volume is determined pre- and postbypass by determination of weight, hematocrit, and hemoglobin. During bypass blood volume is determined by central venous pressure, the status of injection of the conjunctival blood vessels, and blood balance as determined from blood loss in suction and thoracotomy bottles, blood transfusions added, level of blood in oxygenator, and estimated loss in sponges, drapes, towels, and on the operating table. A fairly accurate appraisal of this latter factor of estimated blood loss can be determined from analysis of previous cases and is influenced by duration of bypass, type of procedure, primary or secondary operation, size of patient, and type of incision (midline, unilateral, or bilateral thoracotomy). By so following the above criteria and correcting deviations over 90 per cent of the patients left the operating table with blood volumes within ± 100 c.c. of the preoperative level. Immediate correction of the others was instituted.

Anticoagulation and Reversal Control.—All blood to be used during surgery is collected in siliconized bottles on the morning of operation. The patient's blood cells are typed for various group antigens and all the donors' blood is cross-matched, employing saline conglutin and Coombs' antiglobulin. The blood to be used during bypass for priming and replacement is collected in heparin bottles (15 mg. per 600 c.c.). The blood used following bypass is collected in nonvacuum bottles with acid citrate as the anticoagulant. Just prior to cannulization, heparinization is accomplished by the administration of 0.75 mg. of heparin per pound of body weight. Following removal of the cannulae, protamine (0.5 mg. per pound) is slowly given intravenously. Frequently an additional dose of protamine (one-third to one-half of the original dose) is administered during the first postoperative hour.

Cannulations.—The superior and inferior venae cavae are cannulated through the right auricular chamber. With the use of controlled venous return based upon vena caval pressure and oxygenator blood level, it is advantageous to have both the superior and inferior caval systems monitored and individual caval flow subject to control. The vena caval catheters should be so placed that they lie free in these vessels and should not be introduced by mistake into the innominate, azygous, or hepatic veins. Central venous pressure as taken in the superior vena cava is an important factor not only in controlling and monitoring circulating blood volume but also in the prevention of cerebral edema. Recently, inferior vena caval pressures have also been monitored. As a whole, they tend to reflect superior vena caval pressure but may be affected by more factors. The arterial input catheter has always been placed in the femoral artery through the superficial femoral artery, except for the first 4 patients. The largest Teflon cannula 3½ inches in length that will fit snugly into this vessel is used. An arterial pressure manometer is inserted into the other superficial femoral artery.

Elective Cardiac Arrest.—Elective cardiac arrest has been employed in 45 operations. The Melrose[20] technique introduced clinically by Effler and associates[10] was utilized. Potassium chloride, 10 mEq. for children, 20 mEq. for

adults, diluted in 50 c.c. of arterial blood, was the arresting agent. The amount of solution necessary to stop the heart is injected into the ascending aorta (Fig. 6). Acetylcholine was used in one case.[16]

Our present indications for elective cardiac arrest are (1) lesions obscured by excessive regurgitant blood and (2) lesions, which, because of their location, are difficult to close with the heart beating. These include tetralogy of Fallot and some ventricular septal defects.

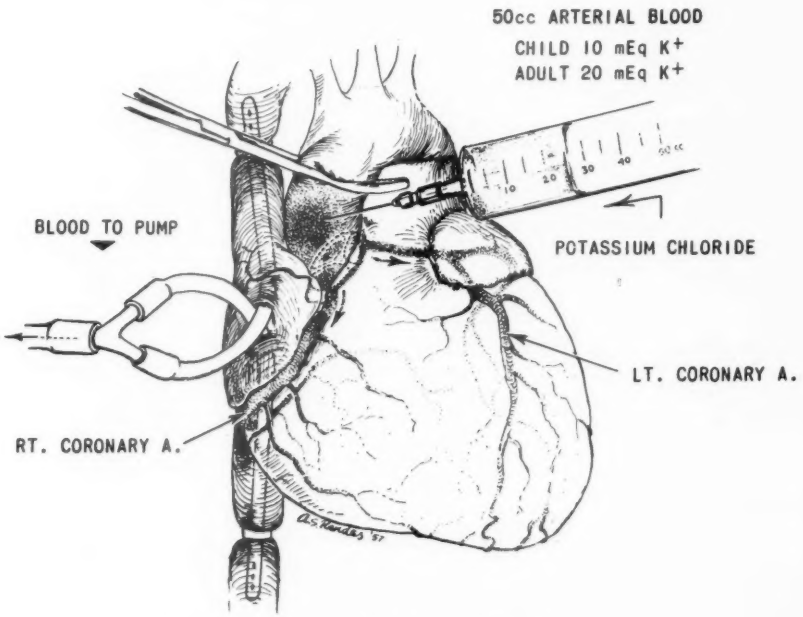

Fig. 6.—Method used to produce elective cardiac arrest.

Ventricular fibrillation in the immediate post-arrest period has occurred in 31 cases (Tables II and III). In 8 cases the heart converted to normal rhythm with continued perfusion. In 13 patients defibrillation with electric shock was successful (0.1 second duration at 60 to 110 volts). Defibrillation was successful in 7 patients with Isuprel (0.1 to 0.2 mg. added to the oxygenator). Three patients had persistent fibrillation with no recovery in spite of repeated attempts at defibrillation and continued perfusion for long periods.

Treatment of Post-arrest Ventricular Fibrillation.—The present treatment of ventricular fibrillation following elective cardiac arrest is:

1. Perfusion is continued and/or increased.

2. Distention of the heart is avoided by manual compression, if necessary. The aortic caval clamps are opened slowly to prevent flooding and dilating the heart.

3. If the blood pressure is low, Aramine (2.5-5 mg.) is added to the oxygenator. The pumps are regulated to increase the patient's blood volume, if it appears low.

4. Oxygenation is increased by raising the disc rotation rate.

TABLE II. DATA ON 45 ELECTIVE CARDIAC ARRESTS

LESION	DURATION OF AORTIC OCCLUSION (MIN.)	POST-ARREST VENTRICULAR FIBRILLATION	TREATMENT
IVSD	13	10 min.	Electric defibrillation 0.1 sec., 110 volts
IVSD	21	None	Spontaneous systole with perfusion
IVSD	18	20 min.	Electric defibrillation 0.1 sec., 110 volts
IVSD	7	None	Spontaneous systole with perfusion
IVSD	23	9 min.	Spontaneous systole with perfusion
IVSD	37	None	Spontaneous systole with perfusion
IVSD	28	1 min.	Spontaneous systole with perfusion
IVSD	15	None	Spontaneous systole with perfusion
IVSD	28	8 min.	Electric defibrillation 0.1 sec., 85 volts*
IVSD	41	24 min.	Electric defibrillation 0.1 sec., 80 volts
IVSD	35	8 min.	Electric defibrillation 0.1 sec., 110 volts†
IVSD	59	None	Spontaneous systole with perfusion
IVSD	11	3 min.	Spontaneous systole with perfusion
IVSD and IASD	24	3 min.	Spontaneous systole with perfusion
Tetralogy	11	6 min.	Electric defibrillation 0.1 sec., 85 volts
Tetralogy	7	2 min.	Spontaneous systole with perfusion
Tetralogy	17	3 min.	Spontaneous systole with perfusion
Tetralogy	26	None	Spontaneous systole with perfusion
Tetralogy	50	18 min.	Recovery with Isuprel, 0.1 mg.
Tetralogy	60	19 min.	Electric defibrillation 0.1 sec., 110 volts
Tetralogy	38	None	Spontaneous systole with perfusion
Tetralogy	23	5 min.	Recovery with Isuprel, 0.1 mg.
Tetralogy	13	2 min.	Electric defibrillation 0.1 sec., 60 volts
Tetralogy	41	4 min.	Electric defibrillation 0.1 sec., 85 volts
Tetralogy	46	2 min.	Spontaneous systole with perfusion
Tetralogy	30	None	Spontaneous systole with perfusion
Tetralogy	60	None	Spontaneous systole with perfusion
Reoperation—tetralogy	16	None	Spontaneous systole with perfusion
Reoperation—tetralogy	35	None	Spontaneous systole with perfusion

*Acetylcholine cardioplegia.

†Ventricular fibrillation to ventricular tachycardia. Treatment, Lanatoside C in the oxygenator.

TABLE II—CONT'D

LESION	DURATION OF AORTIC OCCLUSION (MIN.)	POST-ARREST VENTRICULAR FIBRILLATION	TREATMENT
Reoperation—tetralogy	35	6 min.	Recovery with Isuprel, 0.1 mg.
Pentalogy	54	8 min.	Electric defibrillation 0.1 sec., 60 volts
Pentalogy	21	None	Spontaneous systole with perfusion
Pentalogy	29	16 min.	Electric defibrillation 0.1 sec., 60 volts
Transposition	2	8 min.	Spontaneous systole with perfusion
Coarctation of aorta and IVSD	44	> 1 hour	No recovery‡
Mitral insufficiency	24	> 1 hour	No recovery
Mitral insufficiency	16	17 min.	Recovery with Isuprel, 0.2 mg.
Mitral insufficiency	18	None	Spontaneous systole with perfusion
Mitral insufficiency	35	> 1 hour	No recovery
Mitral insufficiency and aortic stenosis	16	12 min.	Recovery with Isuprel, 0.1 mg.
Mitral stenosis	8	8 min.	Electric defibrillation 0.1 sec., 110 volts
Mitral and tricuspid stenosis	35	11 min.	Recovery with Isuprel, 0.1 mg.
Aortic stenosis	20	None	Spontaneous systole with perfusion
Aortic insufficiency	17	31 min.	Recovery with Isuprel, 0.1 mg.
Aortic insufficiency	29	16 min.	Electric defibrillation 0.1 sec., 110 volts

‡Old infarct.

5. When the heart appears well oxygenated, with good tone, and in rapid fibrillation, electric defibrillation is attempted. However, approximately one minute before this is attempted, a large dose of succinylcholine chloride (60 to 150 mg.) is added to the oxygenator. Low voltage shock at 0.1 second's duration is used first. If unsuccessful, the voltage is increased. Frequently a single shock is followed by effective systole.

6. If ventricular fibrillation appears slow and wormy, and the heart is flaccid, 0.05 to 0.2 mg. of Isuprel is added to the oxygenator. This is frequently followed within 2 minutes by effective systole (Table III).

TABLE III. SUMMARY OF TREATMENT OF POST-ARREST VENTRICULAR FIBRILLATION

Number of elective cardiac arrests		45
Ventricular fibrillation		31
Treatment		
A. Continued perfusion	8	
B. Isuprel defibrillation	7	
C. Electric defibrillation	13	
D. No recovery	3	

Coronary Perfusion.—The complications attendant on the use of elective cardiac arrest during surgery of the aortic valve have led to the use of direct coronary perfusion. This technique was used on 5 patients in this series (Table IV). Following temporary elective cardiac arrest with acetylcholine the aorta is opened, and the coronary ostia are cannulated with specially designed coronary cannulae. The coronary perfusion is then maintained by an auxiliary pump and reservoir drawing blood from the arterial end of the oxygenator.

Coronary perfusion is increased until cardiac contractions appear normal and the electrocardiogram resembles the pre-bypass pattern (Fig. 7). Perfusion has been maintained for periods up to 160 minutes.

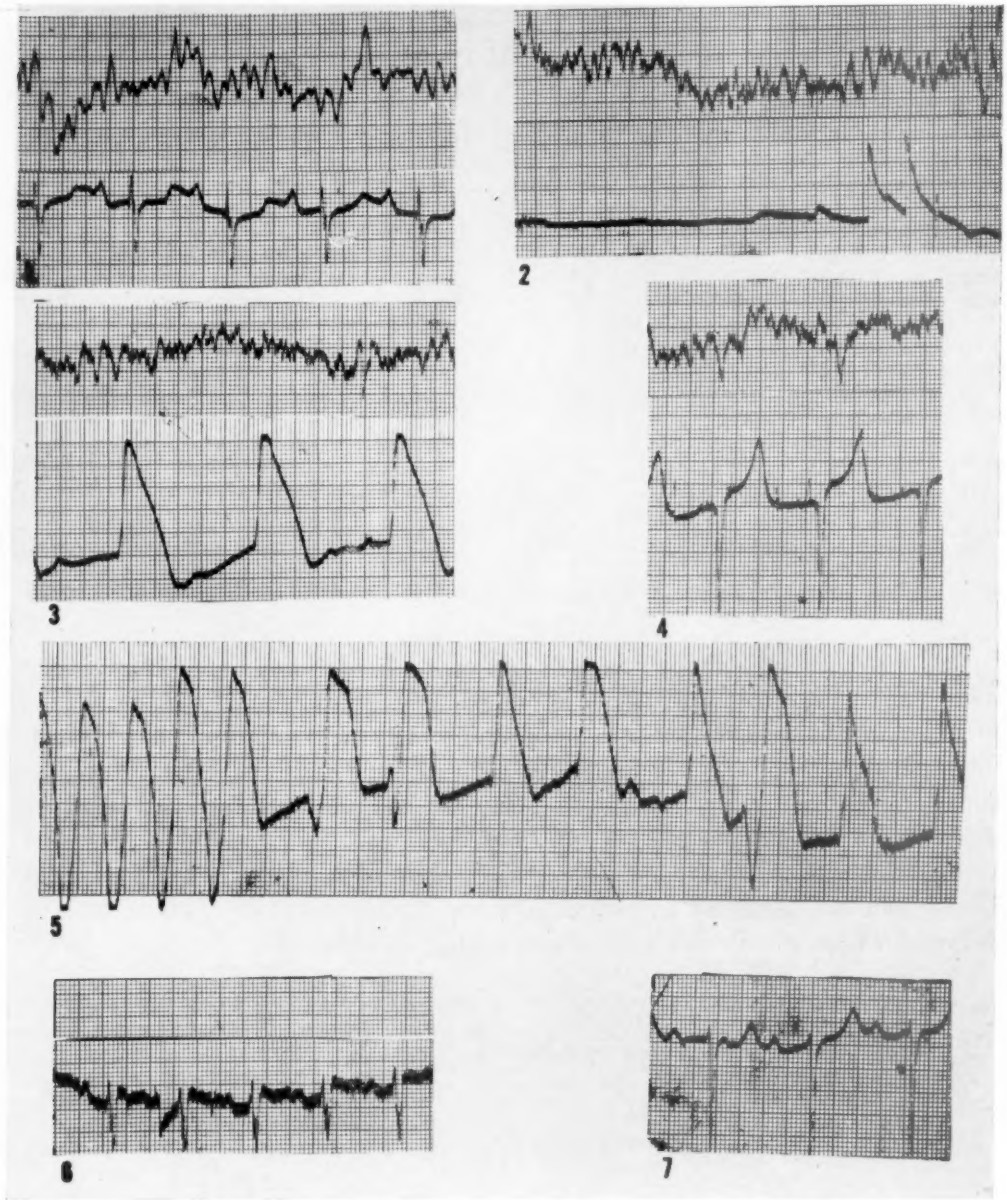

Fig. 7.—Electrocardiogram (Lead II) and EEG of 34-year-old male during total bypass for correction of rheumatic aortic insufficiency. *1*, Prior to bypass. *2*, Acetylcholine arrest. *3*, Coronary perfusion. 250 c.c. per minute. *4*, Coronary perfusion, 300 c.c. per minute. *5*, Idioventricular rhythm terminated by continued perfusion and atropine sulfate, 0.2 mg., into the oxygenator. *6* and *7*, Postperfusion recovery period.

DISCUSSION

The Kay-Cross pump-oxygenator has proved to be a satisfactory means of maintaining circulation and oxygenation during operative repair of intracardiac lesions. The application of direct vision surgery to the repair of

TABLE IV. CORONARY PERFUSION DATA

PATIENT	PERFUSION RATE (C.C./MIN.)	TIME (MIN.)	COMMENTS
A. S.	350	42	Idioventricular rhythm immediately following perfusion Treatment 0.4 mg. atropine
C. C.	200-350	40	Ventricular fibrillation during perfusion Inadequate perfusion*
J. J.	400	72	Adequate perfusion
A. Z.	350	60	Adequate perfusion
W. L.	250-400	160	Adequate perfusion

*Preoperative ECG evidence of coronary disease.

acquired valvular defects has been the result of increased pump-oxygenator capacity, greater knowledge of perfusion techniques, and improvement in operative management.

The monitoring devices used in this series have been valuable adjuncts, not only in operative management of the individual patient, but also in increasing our knowledge of the bypass phenomenon. However, a careful clinical evaluation of the patient's condition remains the best criterion for safe management during operation and especially during the bypass period.

The electrocardiogram has been most useful in determining diagnosis and the results of treatment of arrhythmias, especially following elective cardiac arrest. When direct coronary perfusion is employed, the electrocardiogram, as well as direct visualization, is used as a guide to adequate perfusion.

The electroencephalogram has verified our impression of the depth of anesthesia and the signs of cerebral hypoxia. When the encephalogram has flattened or delta waves have appeared, there has always been simultaneous clinical evidence of inadequate cerebral circulation, that is, widely dilated, nonreactive pupils. This usually accompanies an excessive fall in arterial pressure.

In our hands, the proper operation of the pump-oxygenator depends upon the continuous measurement of femoral artery and superior vena caval pressures. These measurements, together with the clinical evaluation of the patient, that is, the appearance of the conjunctival vessels,[21] enable us to maintain a relatively consistent patient blood volume during bypass.

The Clark oxygen electrode has been a useful qualitative tool in determining what influences pO_2 changes during bypass. We have been unable to verify its accuracy by repeated oxygen saturation determinations with the methods described by Peters and Van Slyke.[24] These determinations were corrected for temperature and pH of the blood.[25] The quantitative inaccuracy of the Clark oxygen electrode, as used in the oxygenator, is probably due to the lack of control of temperature of the electrode and of the agitation rate of blood at the electrode membrane surface, plus the inability to perform frequent calibrations of the electrode. An apparatus has been described by Severinghaus[26] which is designed to eliminate these sources of error.

As has been described by others,[5, 9, 12] metabolic acidosis was observed during bypass. The metabolic acidosis, as evidenced by a fall in blood buffer bases, occurs rapidly after the beginning of bypass (Fig. 5).

The blood used to prime the oxygenator develops a considerable degree of metabolic acidosis. This is in agreement with the findings in earlier work.[21] In 50 consecutive cases shown in Fig. 5, it is seen that the blood buffer base level of the blood in the oxygenator prior to bypass is the same level on the average to which the patient's blood buffer bases fall during bypass. There is no evidence of a progressive fall from this point to the end of the bypass, in a significant number of cases. This will be reported in detail at a later date.

There are two questions which arise: (1) How much of the metabolic acidosis which occurs during bypass is due to the donor blood used to prime the oxygenator? (2) Is the absence of a progressive metabolic acidosis during bypass an indication of adequate perfusion?

In the post-bypass period, there is a spontaneous correction of the metabolic acidosis without specific treatment, provided circulation is adequate. Any metabolic acidosis occurring in the postoperative period is a reflection of the circulatory status of the patient. We have not seen postoperative metabolic acidosis as a primary cause of circulatory failure.[12]

Elective cardiac arrest is a useful and sometimes essential adjunct to open heart surgery. It is a definite added hazard, however, especially in acquired heart disease. It was initially employed during the correction of mitral valvular lesions with the thought that it might be a preventive factor in air embolism. It was subsequently found to be not only unnecessary, but also disadvantageous. The surgical correction of valvular lesions is more difficult to evaluate in the motionless, flaccid heart. The rheumatic heart poorly tolerates elective cardiac arrest, probably because rheumatic heart disease involves the myocardium with scarring, dilatation, and hypertrophy. In aortic valvular disease particularly, the markedly hypertrophied muscle mass may be out of proportion to its available coronary supply. The myocardial anoxia produced by elective cardiac arrest even for short periods leads to difficulty in the post-arrest period, with inadequate systole and/or ventricular fibrillation. This has not infrequently compromised an otherwise successful surgical correction.

The advantages of elective cardiac arrest are (1) ease of closure of septal lesions due to decreased regurgitant blood and a quiet field, (2) reduction in hemolysis due to decreased bypass time and coronary sump suction, and (3) reduction of blood loss during bypass. The disadvantages of elective cardiac arrest are (1) the frequent occurrence of post-arrest ventricular fibrillation, (2) the inability to evaluate the surgical correction of valvular lesions, (3) the possibility of distortion of intracardiac structures, (4) the danger of overdistention of the flaccid arrested heart, and (5) the increased danger of postoperative A-V heart block following the closure of ventricular septal defect. The latter has not been seen in this series, but is reported by others.[1, 4]

The occurrence of post-arrest ventricular fibrillation has been a common complication of elective cardiac arrest (Tables II and III). The flaccid, arrested heart is easily over-distended and this is probably a contributing factor in the etiology of this complication. It is generally agreed that the longer the heart is arrested, the greater the degree of anoxia and possible

myocardial damage which predisposes to the production of ventricular fibrillation. It is difficult to predetermine which heart will tolerate elective cardiac arrest. It is apparent that children with congenital heart lesions, especially those of the cyanotic group, tolerate elective cardiac arrest much more readily than those with acquired lesions.

In patients with ventricular septal defects and tetralogy of Fallot, removal of the aortic clamp and resumption of coronary flow are instituted prior to complete closure of the septal defect, as an aid in the prevention of distention of the left ventricle. In other instances, when necessary, decompression of the left auricle is utilized. These are felt to be valuable adjuncts in restarting the heart and preventing ventricular fibrillation.

Before attempting defibrillation, effort should be made to improve perfusion and oxygenation of the myocardium. During electric defibrillation a marked drop in pO_2 in the arterial end of the oxgenator is noted when no succinylcholine is used. This occurs at a time when adequate oxygenation is of prime importance, i.e., during defibrillation. The massive skeletal muscle contractions occurring with electric shock increase oxygen consumption. This results in a return of grossly desaturated venous blood to the oxygenator.

The administration of a paralyzing dose of succinylcholine chloride into the oxygenator prevents this marked drop in pO_2 as well as the accidental dislodgment of one of the numerous cannulae connected to the patient.

Isuprel has been an effective chemical defibrillating agent when "slow" ventricular fibrillation occurs following elective cardiac arrest. The diagnosis of "slow" fibrillation cannot be made with the electrocardiogram but is made by direct observation of the heart. The ventricles present a wormlike, flaccid appearance.

The mechanism of action of Isuprel in the treatment of ventricular fibrillation is not entirely clear. During ventricular fibrillation the auricles are frequently beating normally. It is postulated that the combination of marked myocardial stimulation, increased conductivity,[17, 22] and stimulation of the higher rhythmic foci produces an impulse which overwhelms the ventricular fibrillation cycle, resulting in ventricular systole.

Direct coronary perfusion permits a safe, prolonged exposure of the aortic valve, with a beating, well-oxygenated heart. Surgery of the aortic valve frequently requires long periods of exposure. It is difficult to predict preoperatively which valves will require extensive valvuloplasty or prosthetic replacement.

The technique of direct coronary perfusion obviates the disadvantages of elective cardiac arrest without coronary perfusion.

The Prevention and Treatment of Heart Block.—Heart block has not been an important factor in this initial series of 200 patients, having actually occurred only once in an instance of surgical correction of transposition of the great vessels 2 years ago. Patients with low atrial septal defects, septum primum, endocardial cushion or atrioventricularis communis defects, tetralogy

of Fallot, and ventricular septal defects are the ones most likely to develop such a complication. When sutures are necessary in areas of the A-V node or bundle, great care is exercised to note electrocardiographic changes during their placement and only small sections of tissue are so included in individual sutures. During the past year, as a precautionary measure, platinum electrodes are sutured to the myocardium and brought out through separate stab wounds, in instances where there has been some transient irregularity in the electrocardiogram during the surgical correction. The electrocardiographic changes noted in open heart surgery have been previously reported.[27]

CONCLUSIONS

1. Modifications of the Kay-Cross pump-oxygenator have increased its clinical application by increasing its oxygenating ability.

2. A number of monitoring devices are used to aid in safe management of open heart surgery. These have supplemented but have not replaced the clinical evaluation of the patient.

3. Ventricular fibrillation is a common complication following elective cardiac arrest, especially in acquired diseases of the heart.

4. A marked drop in oxygen tension in the oxygenator occurs during electric defibrillation. Succinylcholine chloride in paralyzing doses prevents this.

5. Isuprel is an effective chemical defibrillating agent when "slow" ventricular fibrillation occurs.

6. The metabolic acidosis which occurs early during the bypass spontaneously corrects itself in the post-bypass period provided circulation is adequate.

7. Direct coronary perfusion is a valuable adjunct in the management of open aortic valvular surgery.

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SURGICAL CORRECTION OF TRANSPOSITION OF THE GREAT VESSELS

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TRANSPOSITION of the great vessels is a congenital heart malformation which causes cyanosis, with about the same incidence as tetralogy of Fallot. It is one of the most common anomalies which cause death during the first period of life. In complete transposition, the aorta emanates from the right ventricle and receives systemic venous blood. The pulmonary artery emanates from the left ventricle and directs the arterialized blood back to the lungs. The only possibility of survival for children post partum is the presence of associated defects which shunt blood between the systemic and pulmonary circulation. A persistent foramen ovale, an atrial septal defect, a persistent ductus arteriosus, and ventricular septal defect are the most common associated defects and occur in 75 to 100 per cent of the cases reported in the literature.[7, 9]

From a surgical point of view, it can be considered that the pulmonary artery and the aorta have about the same diameter in 60 per cent of the cases. The coronary arteries always leave the aorta and, according to Mustard,[11] they usually extend in front of the pulmonary artery. In 4 of our 6 cases, there were two coronary arteries which originated near each other on the back of the aorta. The right coronary artery was wide and divided into two branches. One right branch corresponded to a normal right coronary artery, and a left branch, extending behind the pulmonary artery, supplied the same region as an ordinary circumflex branch of a left coronary artery. The descending branch of a normal left coronary artery was, in these instances, replaced by a narrow left coronary artery. The right ventricle, maintaining the systemic circulation, is always hypertrophic. The left ventricular myocardium is hypotrophic in cases of intact ventricular septum and closed ductus.

Since the prognosis for these patients is very bad,[4, 7, 9] many attempts have been made to correct transposition of the great vessels. Operative procedures used on the arterial side have been unsuccessful.[2, 3, 8, 11] With operations on the venous side,[4, 6, 9] the survival rate has been improved, but mortality has been high.* Merendino and co-workers[10] have performed interatrial venous transposition with the aid of a plastic prosthesis, with no survivals to the present time. Albert[1] has tried experimentally to use an anterior atrial septal flap for correction.

Two new different surgical methods for complete correction of transposition of the great vessels have been worked out experimentally; one in 1955 on

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*The best results have been those obtained with Baffes' method.[13]

the arterial side, and one in 1957 on the atrial level.[12] The methods have been used clinically, with one survival, by the atrial method. The principle of the experimental work has been to divert the blood flow to or from the ventricles with maintained perfusion pressure in the aorta and the coronary arteries, without using a prosthesis or graft which might result in obstruction of the blood stream later as the children grow.

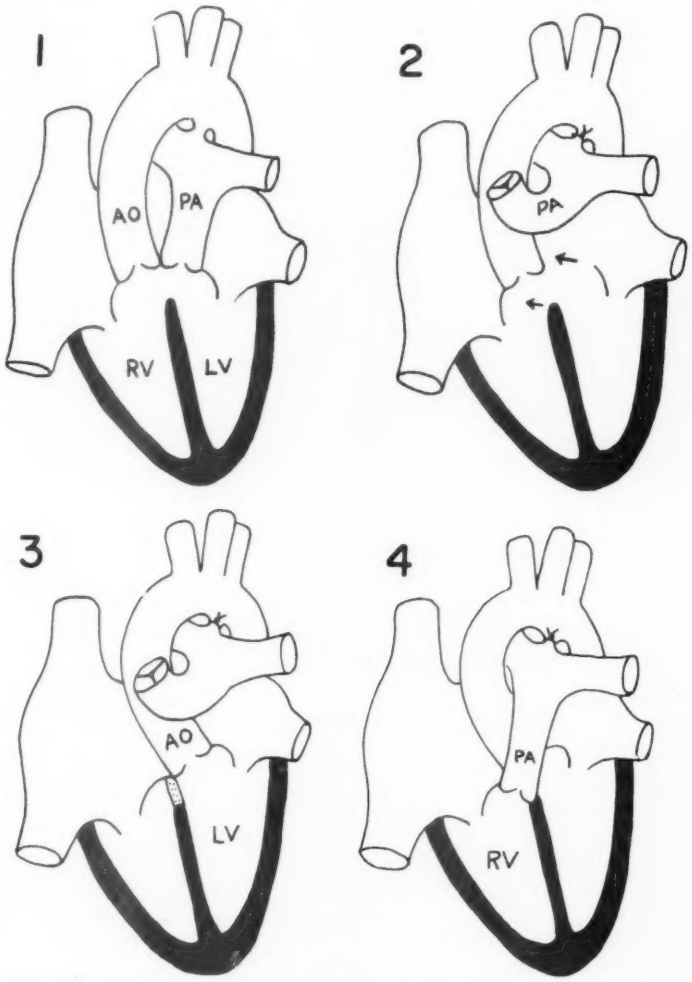


Fig. 1.—Steps in surgical correction on the arterial side of transposition of the great vessels. *1*, Defect in a case of complete transposition of aorta and pulmonary artery combined with ventricular septal defect and persistent ductus arteriosus. *2*, First stage of repair: the pulmonary artery with its valves dissected free; the persistent ductus arteriosus ligated. *3*, Second stage of repair: the root of the aorta sutured to the mitral valve and to the left ventricle; the upper ridge of the ventricular septum sutured to the right of the aortic root. *4*, Repair completed: the root of the pulmonary artery sutured to the right ventricle. *AO*—aorta; *PA*—pulmonary artery; *RV*—right ventricle; *LV*—left ventricle.

CORRECTION OF TRANSPOSITION OF THE GREAT VESSELS ON THE ARTERIAL SIDE

With this method, the aorta, including its coronary arteries, is transposed to the left ventricle and the pulmonary artery to the right (Fig. 1). The pulmonary artery with its valve is dissected from the left ventricle (Fig. 1, *2*). The resulting hole is limited on the right by the aorta and the interventricular

septum and on the left by the anterior mitral cusp, where this meets the atrial wall in annulus fibrosus. This hole is sutured and thus the anterior mitral cusp is fixed to the aortic root (Fig. 1, *2*). A right ventriculotomy is made over the outflow tract. Part of the right aortic border is dissected from the right ventricular wall. An Ivalon sponge prosthesis is sutured into the ventricular septal defect and the upper part of the prosthesis is sutured to the right of the

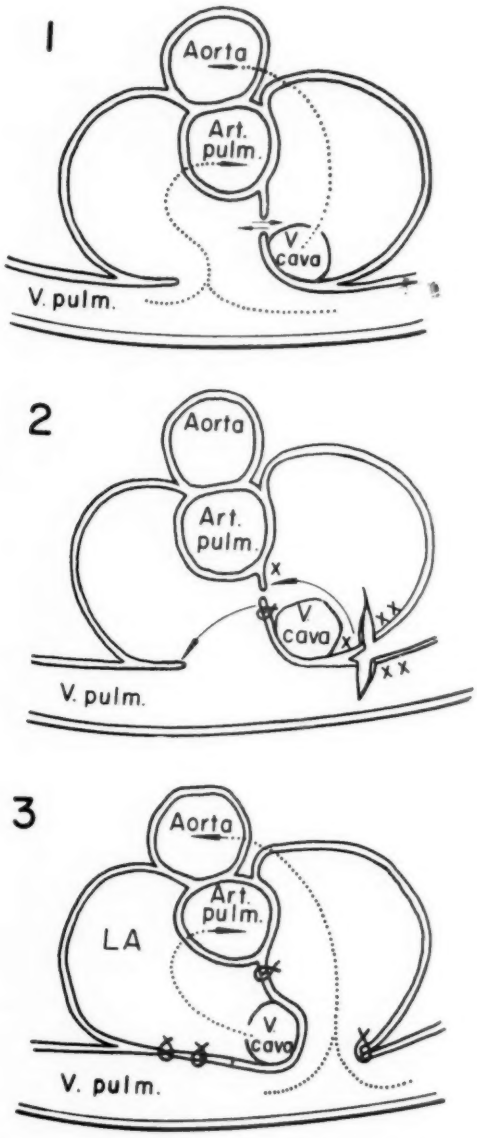


Fig. 2.—Diagram of atrial correction of transposition of the great vessels. *1*, preoperative conditions. *2*, The atrial plastic indicated by arrows. *x* and *xx* indicate incisional edges to be sutured together. *3*, Postoperative condition. Flow pathways indicated.

aorta. Thus, the aorta communicates directly with the left ventricle through the ventricular septal defect (Fig. 1, *3*) and then the pulmonary artery root is sutured into the right ventricle (Fig. 1, *4*). In this way, complete correction of the transposed vessels is achieved.

This method was used in 3 patients, 2 with and one without ventricular septal defect. The first patient, a 6-month-old child, was operated upon under hypothermia (22° C.), combined with perfusion of oxygenated blood through the carotid arteries and induced ventricular fibrillation. During rewarming, after the cardiac correction had been made, there was good cardiac action and the child was breathing spontaneously, when hemorrhage, caused by fibrinolysis, occurred and the baby died.[5]

In 2 cases, operations were performed under extracorporeal circulation with the AGA heart-lung machine. In both these patients, spontaneous heart action was restored with a good pressure in the pulmonary artery during the extracorporeal circulation. However, the patients could not maintain good arterial blood pressure after the bypass was completed and both died.

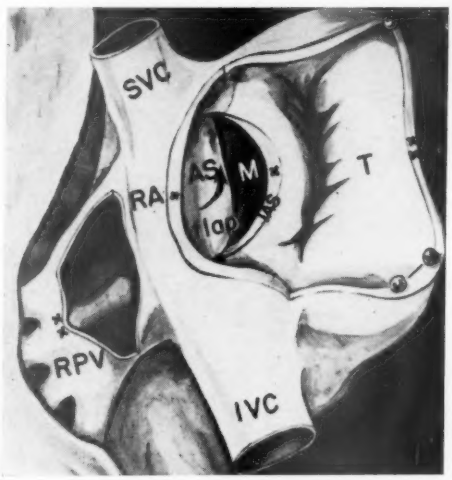


Fig. 3.—Sketch of atrial operation for the correction of transposition of the venous side. Right auricle (*RA*) incised between and in front of venae cavae. Right pulmonary veins (*RPV*) incised at the entrance into the left auricle. Interatrial septum (*IAS*) cut to form a posterior flap (marked with an *arrow*). Tricuspid orifice (*T*), mitral orifice (*M*), superior vena cava (*SVC*), inferior vena cava (*IVC*). The auricularseptal flap will be sutured to the posterior wall of the left auricle in front of the left pulmonary veins, the posterior part of the right auricular wall to the rest of the interauricular septum *x* to *x*, and finally the anterior part of right atrial wall to the right pulmonary veins (*xx* to *xx*).

INTERATRIAL CORRECTION OF TRANSPOSITION

The principle of this operation is illustrated in Figs. 2 and 3.

The operations are performed under extracorporeal circulation with the Crafoord-Senning-AGA heart-lung machine.* A transsternal incision through the fifth costal bed is made. The venous cannulae are introduced into the superior vena cava through the azygos vein, and into the inferior vena cava through the femoral vein. The arterialized blood is returned to the body through the femoral artery. During extracorporeal bypass the aorta is clamped to produce an anoxic cardiac arrest. The clamp is released for a short period every 20 to 25 minutes. If a persistent ductus arteriosus is present, this is ligated. An incision, as long as possible, is made in the right atrium in front of and parallel to the venae cavae. The right pulmonary veins are incised immediately to prevent pulmonary vascular overload; the incision is made as near

*Manufactured by AGA, Lidingö, Sweden.

as possible to the left atrium. The interatrial groove can be slightly dissected to gain some tissue (Fig. 4). By a U-shaped incision, the atrial septum is made into as big a posterior flap as possible. If a large atrial septal defect is present, as in our Case 3, this is widened upward and downward and a posterior flap is formed. The anterior rim of the atrial septum is resected.

When the U-shaped incision is made or the anterior part of the septum is resected, great care must be taken not to damage the A-V bundle and it is therefore safe to leave some tissue intact above and in front of the coronary sinus. In our first 2 patients, the orifice of the coronary sinus was widened into the left atrium by an incision, but in both these patients A-V block occurred.

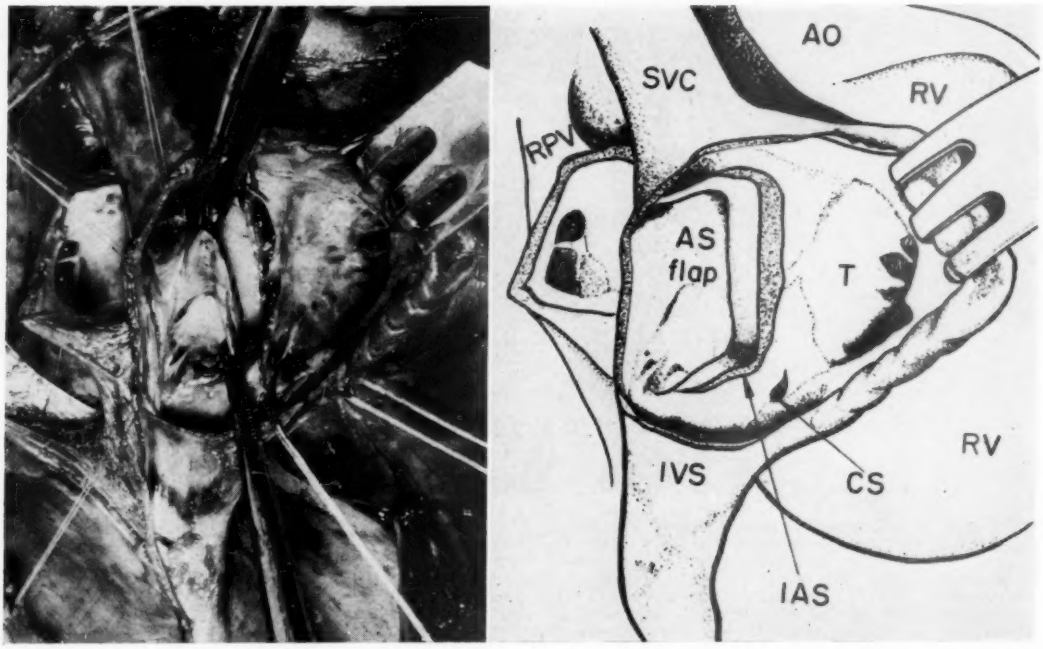


Fig. 4.—Right pulmonary veins (*RPV*) incised along entrance in the left atrium, right atrium incised between, in front of, and parallel to superior vena cava (*SVC*) and inferior venous caval vein (*IVC*). Atrial septum incised creating a posterior flap (*AS* flap, grasped by two forceps). *CS*—Coronary sinus; *T*—tricuspidalis; *RV*—right ventricle; *IAS*—rest of atrial septum. (Postmortem specimen from Case 4.)

Separation of pulmonary veins from the left atrium is achieved by suturing the atrial septal flap to the posterior wall of the left atrium in front of the left pulmonary veins. With three stay sutures placed from the outside of the left atrium, one above, one below, and one between the pulmonary veins and in front of them, the upper corner, the middle part, and the lower corner of the created atrial septal flap are fixed in position. Thereafter, the edge of the atrial septal flap is sutured to the left atrial wall with a continuous over-and-over suture. To get a tight suture line, a finger or an instrument is held behind the flap. In Fig. 5 the suture is completed and Fig. 6 shows the orifices of the left pulmonary veins separated from the anterior part of the left atrium.

Diversion of caval and coronary sinus venous blood to the left and creation of the "venous atrium" are shown in Fig. 7. The anterior edge of the posterior part of the original right atrial wall is fixed to the lower rim of the created atrial septal defect, downward to the anterior and inferior lip of the coronary sinus orifice and then anterior to the entrance of the inferior vena cava. The

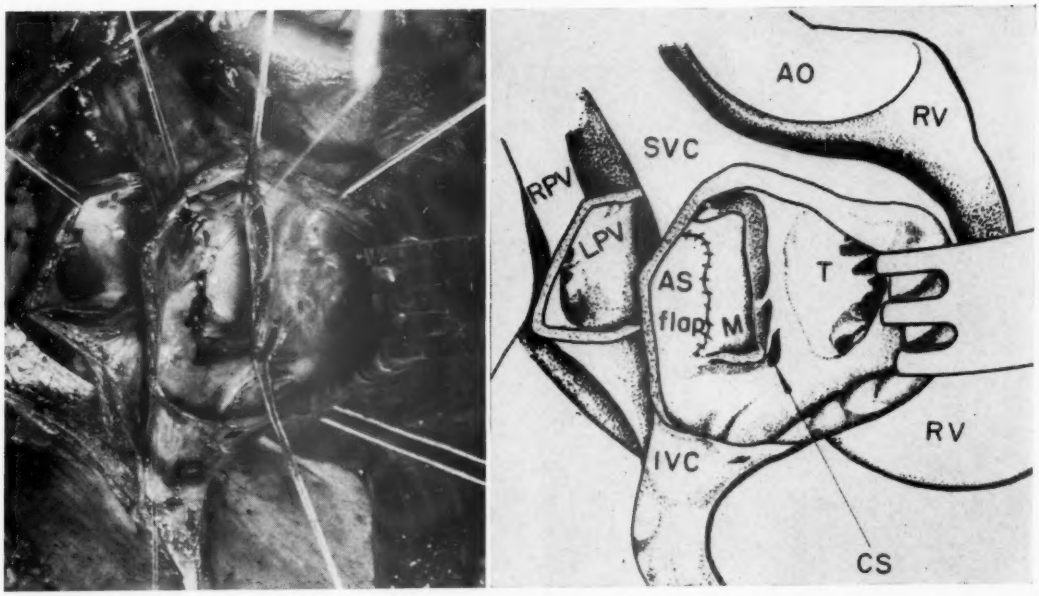


Fig. 5.—Interatrial septal flap sutured to posterior wall of left atrial wall in front of left pulmonary veins. (Postmortem specimen from Case 4.)

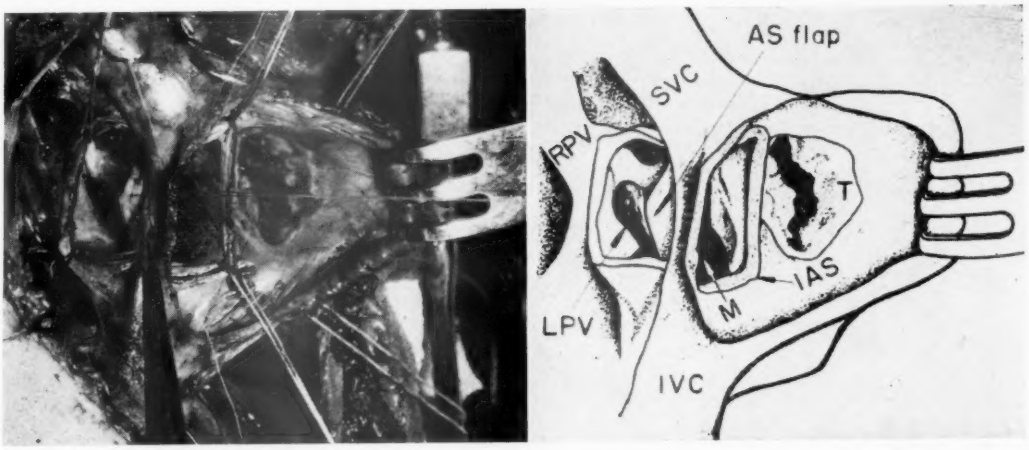


Fig. 6.—The same step as in Fig. 5. Heart rotated forward. The atrial flap is separating the pulmonary veins from the anterior part of the left atrium. (Postmortem specimen from Case 4.)

suture is continued upward over the defect to the posterior wall of the right atrium in front of the superior vena cava. The venous return from the caval veins as well as the coronary sinus blood thus is directed to the left. A "venous

atrium'' is created, limited posteriorly by the atrial septal flap, to the right by the posterior part of the right atrial wall, and anteriorly by the remainder of the left atrium.

Creation of an ''arterial atrium'' is shown in Fig. 8. The pulmonary venous return is shut off from the ''venous atrium'' by the atrial septal flap.

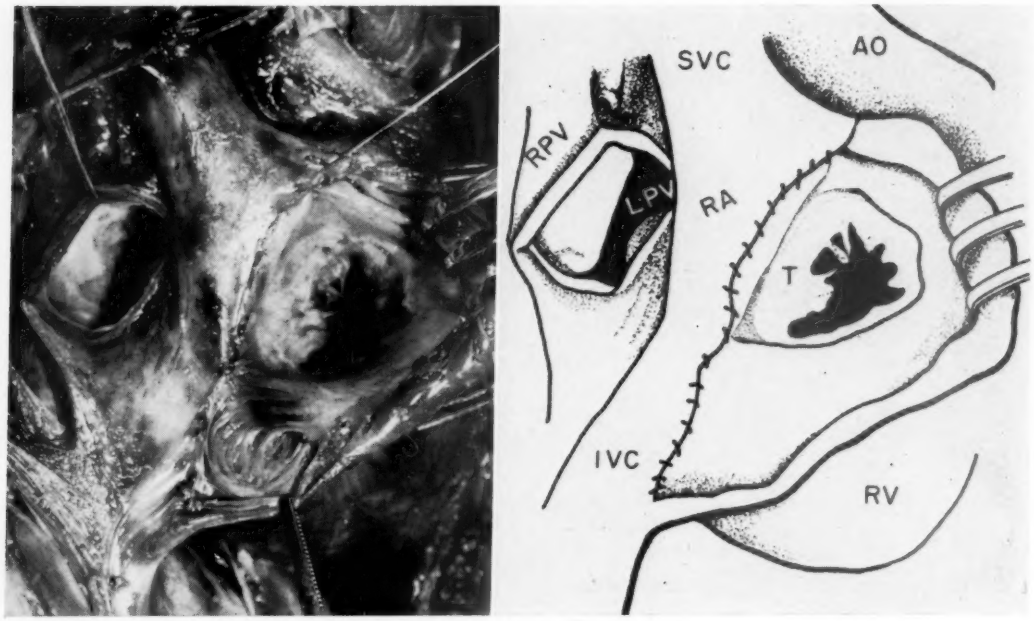


Fig. 7.—Creation of a "venous atrium" by diversion of caval and coronary sinus venous blood to the left. The posterior part of the lateral right atrial wall (*RA*) sutured anterior to and to the right of the inferior vena cava and the coronary sinus, further into the created interatrial septal defect and anterior to the superior vena cava. A "venous atrium" is created consisting of the anterior part of the left atrium limited posteriorly by the atrial septal flap and to the right by the posterior part of the right atrial wall. Into this venous chamber both caval veins and coronary sinus empty. (Postmortem specimen from Case 4.)

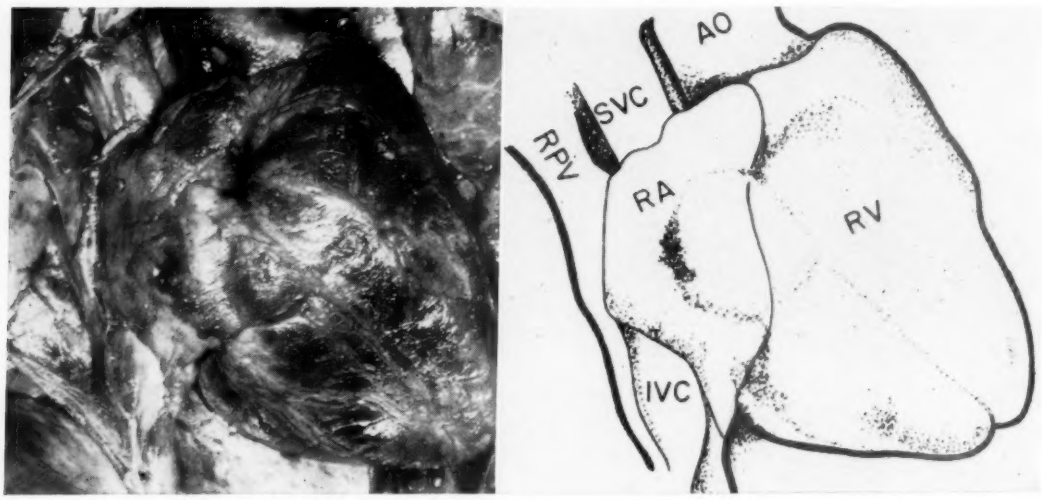


Fig. 8.—The creation of an "arterial atrium." The edge of the anterior rest of the right atrial wall is sutured anteriorly to the wall of both caval veins and to the lateral edge of the incision in the right pulmonary veins. The "arterial atrium" consists of the posterior part of left atrium (with the entering pulmonary veins) and the anterior part of the original right atrium. (Postmortem specimen from Case 4.)

The "arterial atrium" is created by suturing the edge of the anterior part of the right atrial wall, first in front of the entrance of both caval veins into the original right atrium without strangulating the entrance, and then to the lateral incisional edge of the right pulmonary veins. The "arterial atrium" consists of the posterior part of the original left atrium with the entrance of

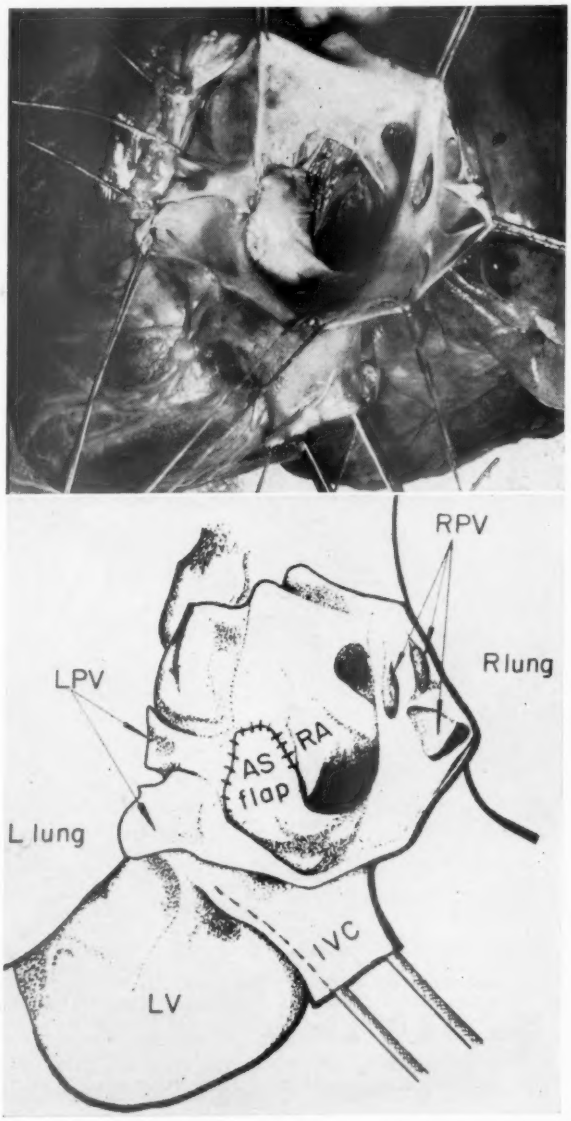


Fig. 9.—"Arterial atrium" opened and seen from behind. Forceps into the "venous atrium" through inferior caval vein. The posterior part of the original left atrium with entering pulmonary veins empty through a wide opening into the anterior remainder of the original right atrium. (Postmortem specimen from Case 4.)

the pulmonary veins, limited anteriorly against the left auricle by the atrial septal flap and communicating widely behind the venae cavae with the anterior remainder of the original right atrium.

Figs. 9 and 10 are shown to clarify the condition of the created atrial chambers. In Fig. 9, the "arterial atrium" with the pulmonary veins is opened

from behind with a transverse incision. A pair of forceps is introduced into the "venous atrium" through the inferior vena cava. The atrial septal flap separates the vena cava from the "arterial atrium." The wide communication between the pulmonary veins and the anterior remainder of the original right atrium is seen.

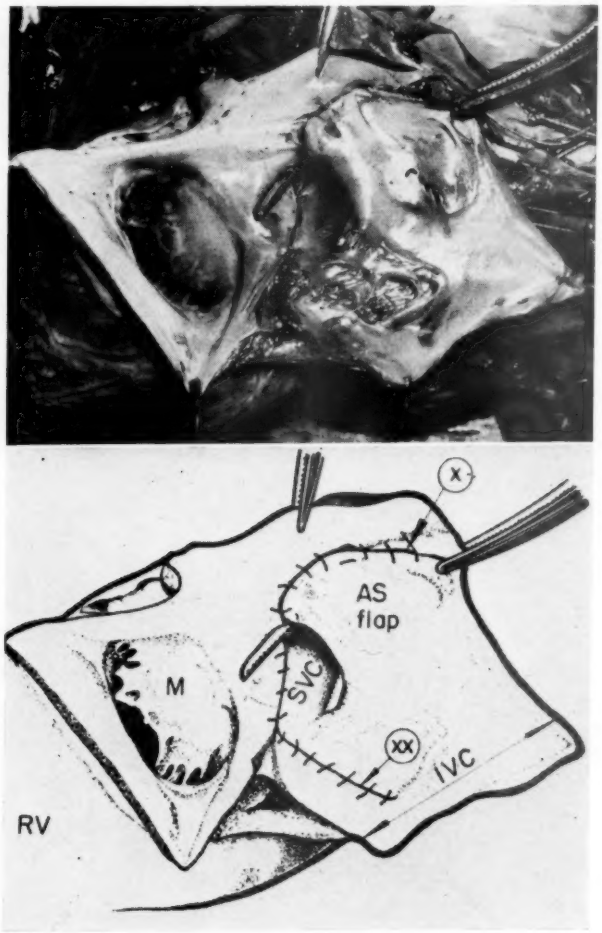


Fig. 10.—"Venous atrium" seen from behind, opened through the inferior vena cava (along incision indicated in diagram of Fig. 9). The posterior wall is folded upward. Forceps in the orifice of the superior vena cava. Suture line between posterior left atrial wall and atrial septal flap is seen above forceps. *x*—Suture line between posterior part of the right atrial wall and the created atrial septal defect below (*xx*). Orifice of coronary sinus hidden. (Postmortem specimen from Case 4.)

In Fig. 10 the "venous atrium" is opened along the line indicated in the diagram of Fig. 9. The posterior wall is folded upward and grasped with two clamps. The forceps are inserted from above into the orifice of the superior vena cava. The suture line between the atrial septal flap and the posterior wall of the left atrium is seen above the forceps. The suture line of the posterior part of the lateral wall of the original right atrium, sutured into the created defect to divert venous blood to the left, is seen below the forceps. There is no strangulation of the entrance of the venae cavae into the "venous atrium." (The orifice of the coronary sinus is masked in the photo.)

CASE REPORTS

CASE 1.—A 6-week-old child, severely ill, had a diagnosis of transposition plus persistent ductus arteriosus plus small open foramen ovale. The arterial oxygen saturation was 39 per cent. Ventricular fibrillation occurred before cannulation was finished. Extracorporeal circulation was started. Pulmonary vascular overload with pulmonary edema was caused by flow through a wide typical persistent ductus arteriosus before the ductus could be dissected and clamped. A typical operation was performed. After correction under anoxic cardiac arrest and release of the aortic clamp, spontaneous defibrillation occurred. There was good heart action, but complete A-V block. There was 94 per cent arterial oxygen saturation when the patient was ventilated with 100 per cent oxygen. The child was awake for a few hours. There was increasing respiratory resistance and ventilatory insufficiency developed. The patient died 18 hours postoperatively. Postmortem examination showed complete surgical correction. The lungs were engorged with blood and edematous fluid. Cause of death: pulmonary vascular overload.

CASE 2.—The patient was a boy, 10 years of age, who was cyanotic and underdeveloped. The diagnosis was transposition of great vessels plus ventricular septal defect and valvular pulmonary stenosis. There had been cerebral emboli with hemiplegia at 2 years of age. The arterial oxygen saturation was 62 per cent. The pressures in both ventricles were equilibrated. The operation was a typical atrial correction of transposition of great vessels plus closure of the open foramen ovale plus closure of a ventricular septal defect with insertion of an Ivalon sponge prosthesis and resection of left ventricular subvalvular stenosis plus pulmonary valvular commissurotomy. During the operation, the left ventriculotomy was first sutured directly without resection of the left ventricular outflow tract, but the resistance to the left ventricle was found to be too great. For this reason the ventriculotomy was reopened, the resection made, and the ventriculotomy closed again. Since the ventricular septal defect was closed during this procedure a pulmonary overload with pulmonary edema developed rapidly. The patient died from increasing ventilatory insufficiency 5 hours postoperatively.

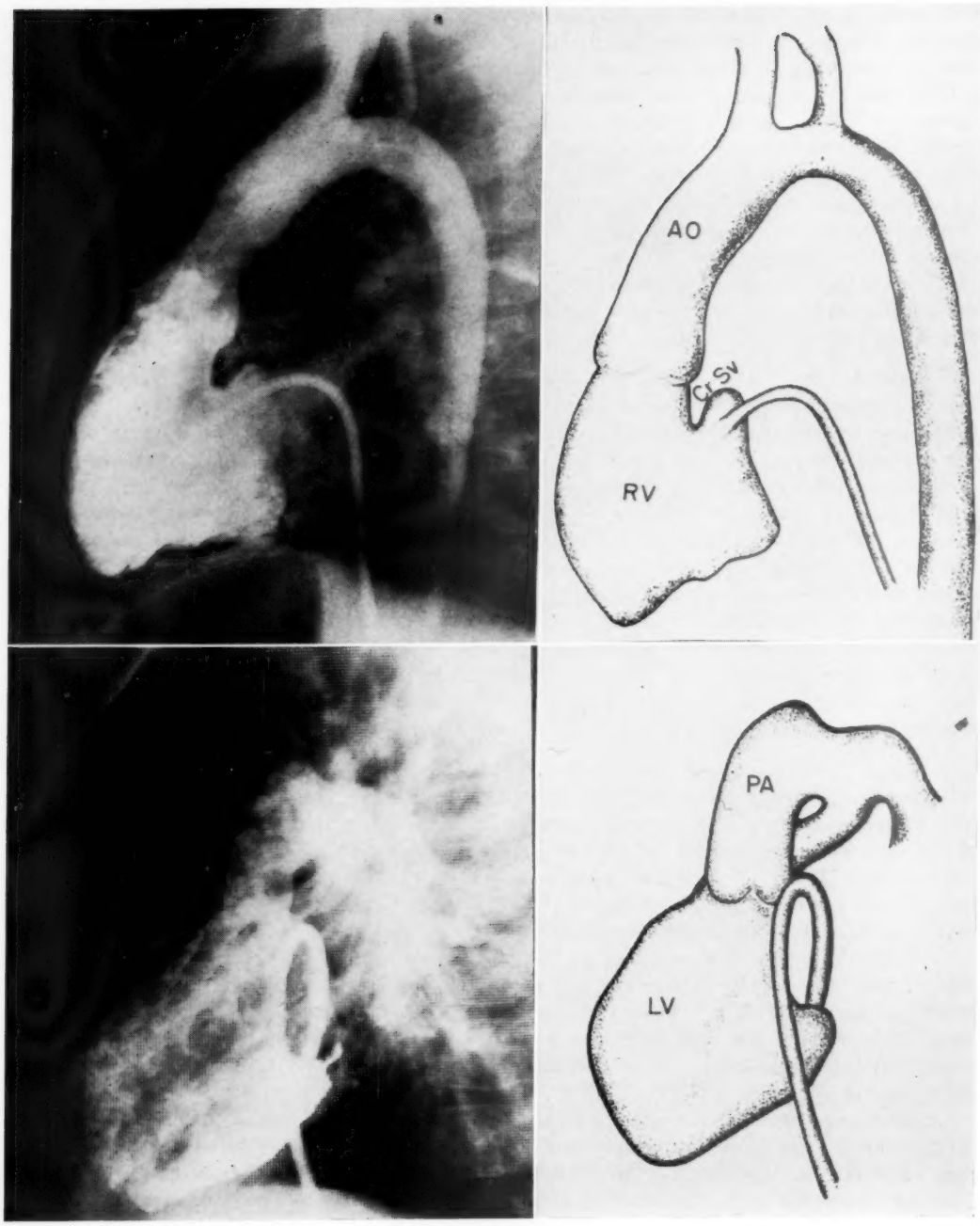
CASE 3.—The patient was a 9-year-old boy with cyanosis. The diagnosis was transposition of the great vessels and atrial septal defect. The arterial oxygen saturation was 69 per cent. Selective angiocardiography with injection of dye into the right ventricle showed the aorta originating from an anatomically typical right ventricle with hypertrophic myocardium (Fig. 11, *A*). The operation was a typical one for atrial correction and ligature of persistent ductus arteriosus under anoxic cardiac arrest. The anterior rim of the atrial septum was resected and the posterior part of the atrial septum used as flap.

After correction and releasing of the aortic clamp, good spontaneous heartbeats with regular auricular and ventricular action were rapidly restored and there was good arterial blood pressure. After the bypass was finished, arterial saturation was 100 per cent on ventilation with 60 per cent oxygen. A tracheostomy was performed and the patient was under artificial ventilation in a respirator for 3 days. There was an uncomplicated postoperative course.

Re-examination 6 weeks postoperatively showed no cyanosis, and catheterization showed no signs of stenosis of the entrance of the venae cavae into the created left "venous atrium," and no shunts on the atrial or ventricluar level. The arterial saturation was 94 per cent (during anesthesia and spontaneous respiration). The patient was in extremely good physical condition.

Angiocardiography with injection into the venous atrium (Fig. 12, *A* and *B*) showed normal reflux into both superior and inferior venae cavae and no stenosis. The left ventricle and pulmonary artery were filled from the malformed "venous atrium." During the refilling phase (Fig. 12, *C* and *D*), the pulmonary veins are seen separated from the remainder of the left atrium forming together with the remainder of the original right atrium the new "arterial atrium." There were no shunts. These conclusions are confirmed on side views (Fig. 13).

A.



B.

Fig. 11.—Preoperative selective angiocardiograph.

A, Injection in right ventricle. Aorta emanates from an anatomically typical right ventricle. *CrSu*—Crista supraventricularis.

B, Injection in left ventricle. The pulmonary artery emanates from an anatomically typical left ventricle.

(Angiocardiography was performed in the Department of Radiology of the Children's Clinic and Thoracic Clinic, Karolinska Sjukhuset.)

CASE 4.—The patient was a 6-year-old underdeveloped girl with cyanosis. The diagnosis was transposition of the great vessels and ventricular septal defect. The arterial oxygen saturation was 59 per cent. A typical atrial correction was made during 17 minutes of extracorporeal circulation. The aorta was unclamped and there was good regular heart action with regular atrial action and no A-V block. The extracorporeal circulation was interrupted and there was good atrial blood pressure. Extracorporeal circulation was started again and the aorta was clamped. An Ivalon sponge prosthesis was sutured into a 1 by 1.5 cm. wide ventricular septal defect through a left ventriculotomy followed by a complete A-V block with low ventricular frequency and low arterial blood

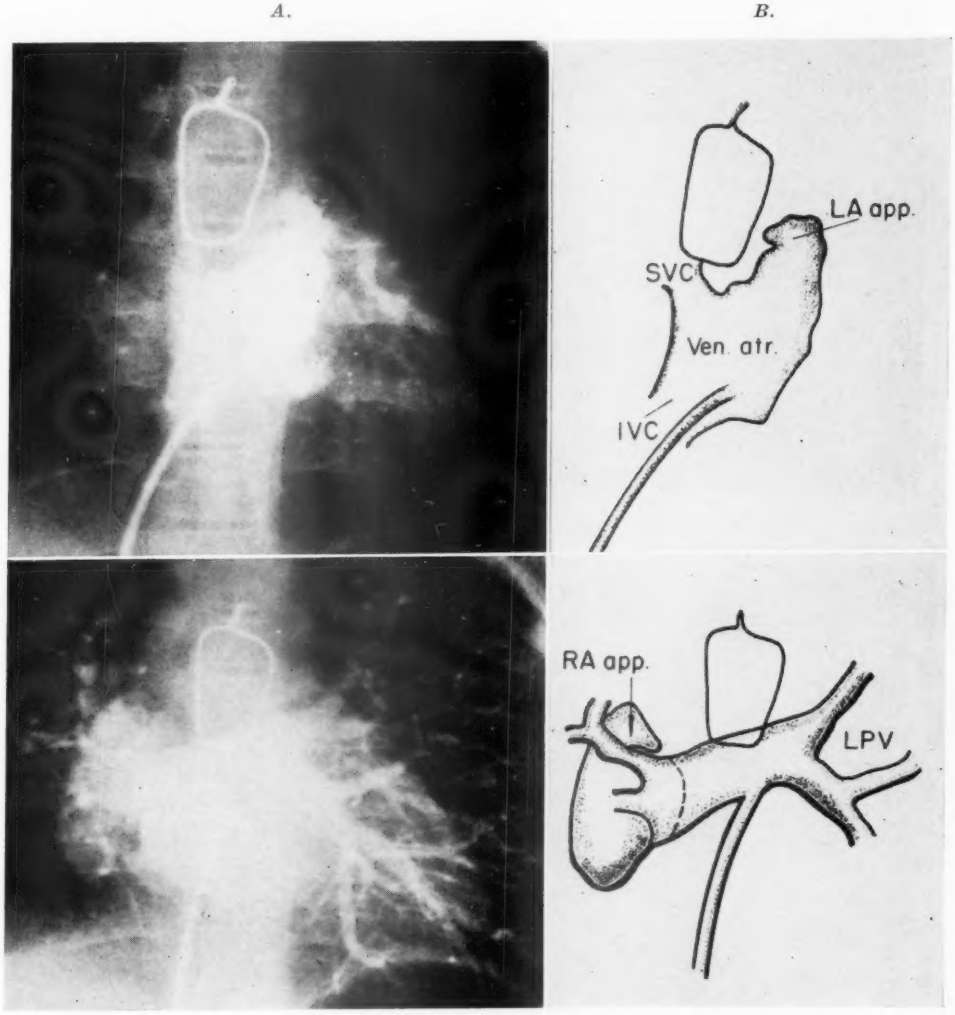


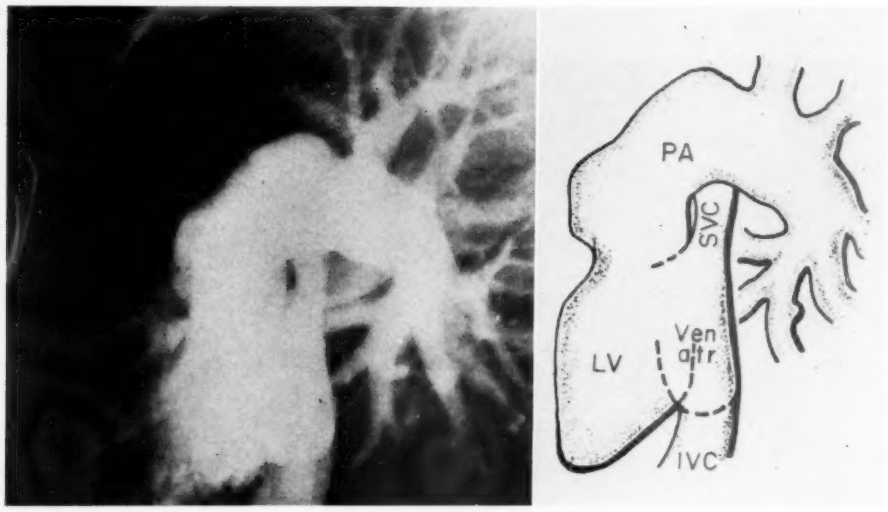
Fig. 12.—Postoperative angiocardiography. Injection into "venous atrium." Frontal projection. *A* and *B*, "Venous atrium" filled. Reflux into caval veins. *LA app.*—Left atrial appendage. *C* and *D*, "Arterial atrium" in pulmonary refilling phase. *RA app.*—Right atrial appendage.

pressure. With Adrenalin the A-V block changed to 1:2, with higher frequency and improved blood pressure. It was difficult to maintain good blood pressure. The patient was conscious for 4 hours. The arterial oxygen saturation was 97 per cent. The blood pressure fell and death ensued. On removal of the drainage tubes, about 400 c.c. of blood poured out from

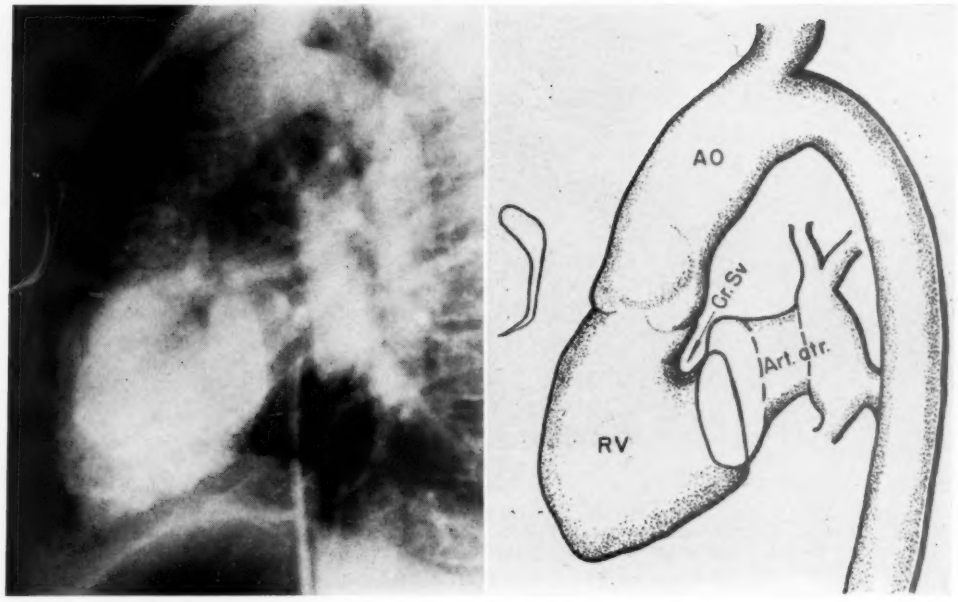
the right chest. A postmortem examination showed another 200 c.c. of blood in the chest cavities. The heart showed complete atrial correction (Figs. 4-10). The ventricular septal defect was well closed. The contributing cause of death was intrathoracic bleeding.

DISCUSSION

Transposition of the great vessels is a congenital anomaly with a bad prognosis. Since operations for this anomaly have to be performed in small children, a method for correcting the defect has been developed avoiding the



A.



B.

Fig. 13.—Postoperative angiocardiography. Injection into "venous atrium." Side projection.

A, Caval veins, "venous atrium" (*Ven. atr.*), left ventricle, and pulmonary artery visualized.

B, Refilling phase. "Arterial atrium" (*Art. atr.*), right ventricle, and aorta visualized.

use of prosthesis or grafts which later, when the child is growing, may lead to obstruction of the blood stream.

The described method used on the arterial side for correcting transposition was used clinically three times. In all 3 cases there was a good postoperative blood pressure in the pulmonary artery, but in the last 2 cases the pressure in the aorta was very low.

Before the operation, the left ventricle had mainly supplied the pulmonary circulation; however, as in 2 of the cases there was a large ventricular septal defect, the left ventricle should have been sufficiently strong to be able to maintain a good systemic circulation. Possibly there was a mitral or aortic insufficiency due to valvular distortion, resulting from the suture of the hole left after the dissection of the pulmonary artery root. Whether or not there were other causes of death is difficult to judge. This method seemed suitable in cases with large ventricular septal defects but as the surgical technique is rather difficult, no further clinical trials have been made.

Methods for correcting transposition on the atrial level were worked out. Principally, the original atrial chambers are rebuilt to form a "venous atrium" consisting of the anterior part of the original left atrium together with the posterior part of the right atrium. The "arterial atrium" consists of the posterior part of the left atrium with the entrance of pulmonary veins and the anterior part of the right atrium.

There are several procedures to be considered; pulmonary veins can be separated from the left atrium by a duplication of the posterior wall or with the aid of a small prosthesis, or part of the right atrial wall. The venae cavae can be diverted to the left through a created atrial septal defect with the aid of an anterior atrial septal flap or a small prosthesis. The atrial septum can be incised longitudinally, a posterior flap being used to divert pulmonary venous return and an anterior flap to divert systemic venous return.

According to electrocardiographic examination, catheterization, and angiocardiography of the surviving patient, the function of the atrial chambers seems to be good despite this remodeling. In this patient, there are no arrhythmias; the different parts of the atrium seem to contract simultaneously. Catheterization reveals no stenosis between the caval veins and the venous atrium and, on angiocardiography, the reflux to the superior and the inferior venae cavae seems to be normal.

In the first 2 cases, the coronary sinus was incised from the left atrium to facilitate diversion of the coronary sinus blood to the left. This might have been the cause of the A-V block in these 2 patients. In the last patient, the A-V block appeared after closure of the ventricular septal defect.

It is difficult to judge whether or not associated defects should be closed during the same operation. A persistent ductus arteriosus is easily closed and this should be done unless there is a distinct contraindication, for example, excessive pulmonary vascular resistance. An associated ventricular septal defect was closed in Cases 2 and 4. This considerably prolonged the perfusion time and increased operative cardiac trauma and seems to have been the actual cause of death in Case 2, in which a pulmonary vascular overload occurred during closure of the left ventriculotomy. It would evidently have been wise

to postpone the final closure of the "arterial atrium." If there is a ventricular septal defect combined with pulmonary stenosis with left-to-right shunt through the ventricular septal defect (as in Case 2), the patient will be cyanotic even if the transposition is corrected on the atrial level. In these cases, it is necessary to make a left ventriculotomy to release the left ventricular outflow stenosis. The ventricular septal defect should be closed at the same time to avoid the risk of pulmonary vascular overload.

In the event of further cases of associated ventricular septal defect we intend not to close the ventricular septal defect during the first operation unless this can be done through the tricuspid orifice without disturbing the septal cusp of the tricuspid valve. If necessary, the ventricular septal defect can be closed during a subsequent operation.

The successful outcome of the atrial correction of transposition in the third case and the findings from re-examinations indicate that the method described here would provide a practical surgical approach to complete transposition of the great vessels.

SUMMARY

1. Two new methods for complete repair of transposition of the great vessels are presented.

2. One method applied on the arterial side has been tried clinically in 3 cases without success.

3. One atrial method for complete transposition without application of prosthesis or grafts was used in 4 patients.

4. One with a successful correction was re-examined. Ideal circulatory conditions were observed.

5. Closure of associated defects is discussed.

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"MYXOMA" OF THE LEFT ATRIUM SIMULATING PURE MITRAL INSUFFICIENCY

Report of a Case With Successful Removal

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IN THE past 4 years a great many publications have dealt with myxomas of the left atrium.[3, 5, 6, 11-13, 17, 19, 21-25, 27, 29, 33-35, 37, 42, 44, 45, 48, 49] It is somewhat surprising that so much attention has been focused on so rare a subject. Although myxomas comprise approximately 50 per cent of all cardiac tumors, the total incidence of neoplasms of the heart ranges between 0.0017[47] and 0.5 per cent[39] of all neoplasms. Undoubtedly this interest has been due to improved diagnostic and operative methods, making it possible for the first time to diagnose intracardiac tumors during life and to remove them surgically. As a matter of fact, Harvey,[22] in 1957, was prompted to the statement: "Myxoma of the left auricle is now a curable lesion."

Thus far, to the knowledge of the authors, 8 myxomas of the left atrium have been successfully removed. In almost all of the more than 150 reported cases of myxoma of the left atrium, the tumors simulated mitral stenosis, although in a few instances there appeared to be a combination of mitral stenosis and mitral insufficiency. It is the purpose of this paper to report a successfully excised myxoma which had particular significance because it closely mimicked pure mitral insufficiency.

CASE REPORT

N.J.H. No. 12948. The patient was a 22-year-old white man. His first hospital stay was from July 13, 1957, to Aug. 10, 1957. The patient had been asymptomatic until 1955, when he was told, during a Navy examination, that he had a heart murmur. In September, 1955, he developed fatigue, shortness of breath, and fever. He was hospitalized elsewhere and treated with massive doses of penicillin, streptomycin, and oxytetracycline hydrochloride for one month. Blood cultures at that time were negative. He was discharged with an elevated sedimentation rate and kept on prophylactic penicillin. In March, 1956, he was digitalized because of increased dyspnea and, at the same time, prednisone was started because of the increased sedimentation rate. At the time of his first admission, a diagnosis of mitral insufficiency was made. There was no sign of cardiac failure and the patient's symptoms remained well controlled on gitalin, 0.5 mg. daily, and a low-salt diet. The erythrocyte sedimentation rate rose to 32 mm. in 60 minutes; the C-reactive protein was 3 plus, and the mucoproteins were 7 units. A right heart catheterization was attempted, but the catheter could not be passed through the tricuspid valve. It was felt that the change in the protein fractions was due to prednisone withdrawal, suggesting a chronic type of active rheumatic fever. Consequently, the patient was discharged on prednisone, 10 mg. daily, and acetylsalicylic acid, 45 grains

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daily, the therapy to continue for at least 3 months. One month prior to his second admission, he developed an illness characterized by general malaise, aching, low-grade fever, and precordial pain. Because of this, he was readmitted on March 14, 1958.

Physical Examination.—The blood pressure was 100/70 mm. Hg, pulse rate 85 per minute, regular. Dyspnea was present at rest, but there was no edema or cyanosis. The liver was one fingerbreadth below the right costal margin and not tender. Neck and hand veins were not distended. The precordium was active with prominence of the left thoracic cage. Palpation revealed evidence of a left ventricular thrust. The point of maximal impulse was felt in the fifth intercostal space outside the midclavicular line. On auscultation, a Grade III, high-pitched blowing, holosystolic murmur was audible over the entire precordium, best heard at the apex and well transmitted to the left axilla. The second heart tone was finely split and accentuated. A third heart tone was present and well heard at the apex. A systolic click or extracardiac sound was heard immediately after the first pulmonic sound. The pulse revealed a very prominent catacrotic notch.

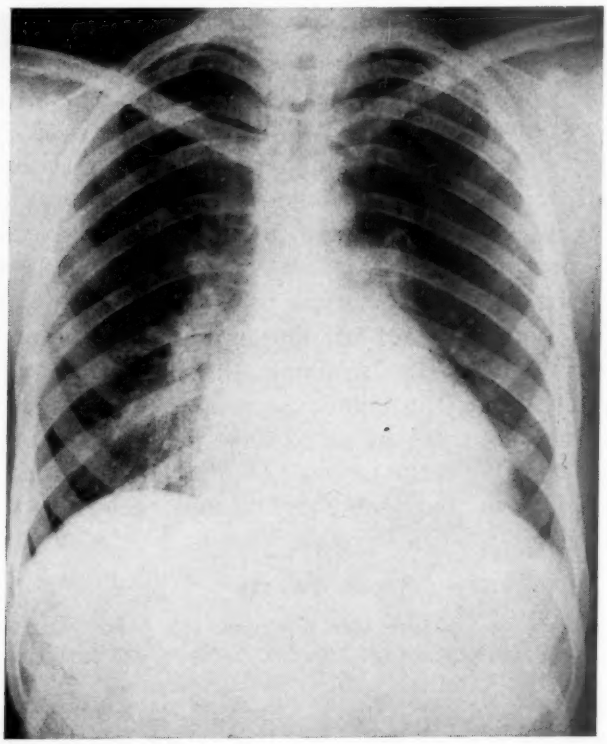

Fig. 1.—Preoperative chest film showing marked enlargement of the left atrium and prominence of the left ventricle.

Course in Hospital.—Fluoroscopy revealed the over-all heart size to be moderately enlarged, with prominence of the left ventricle and marked enlargement of the left atrium (Fig. 1). The pulmonary artery was not remarkable. The vascularity of the lungs was normal. The electrocardiogram revealed left ventricular hypertrophy (Fig. 2). Urinalysis and hematogram were within normal limits. Blood chemistries: fasting blood sugar, 94 mg. per cent; blood urea nitrogen, 14.4 mg. per cent; sodium, 138 mEq.; potassium, 4.25 mEq.; total proteins, 8.2 Gm. per cent; albumin, 5.0 Gm. per cent; globulin, 3.2 Gm. per cent.

Combined right and left heart catheterization was performed on March 21, 1958. The pulmonary artery pressure averaged 88/33, right ventricular pressure 79/0, and right atrial pressure 4/0. A Cournand needle was introduced into the brachial artery

and the pressure recorded as 113/17. The oxygen saturation in the right pulmonary artery at rest was 59 per cent and in the brachial artery was 87 per cent. The cardiac output calculated on the basis of these data was 4.1 L. per minute. The left atrial pressure showed very clearly the a, c, and v waves, and the pressure values, respectively, were 12, 17, and 23.5 mm. Hg. The end diastolic pressure was 9 mm. Hg. The pressure in the left ventricle was 101.5/4 mm. Hg.

The conclusion was that the high mean left atrial pressure showed an obstruction at the level of the mitral valve, and from the atrial pressure tracing there was clear evidence that this obstruction was due to mitral insufficiency. There was no sign of mitral stenosis. Also, the rather high pulmonary hypertension could be explained on this basis. The impression was that mitral insufficiency with secondary pulmonary hypertension was present.

Operation.—The patient was presented to the Cardiac Surgical Conference, where it was decided that the mitral insufficiency should be repaired by the Lillehei modification of the Nichols plication.

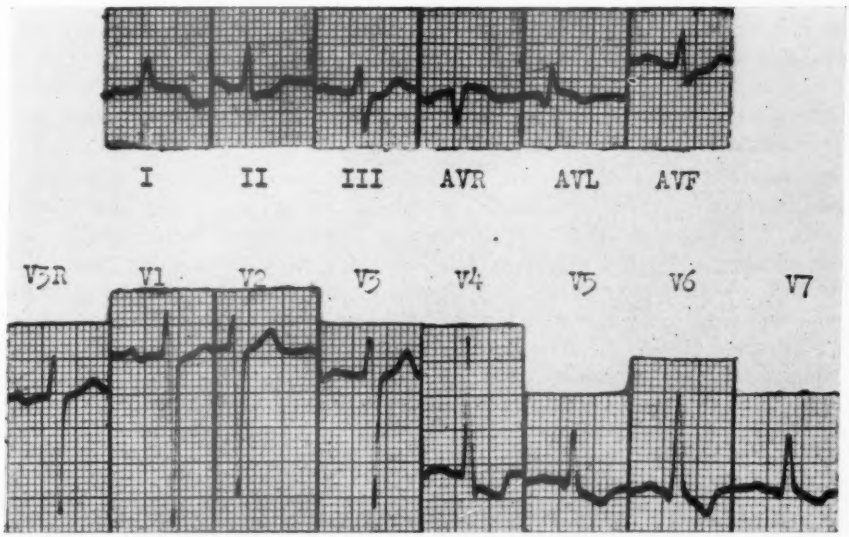

Fig. 2.—Preoperative electrocardiograms revealing left ventricular hypertrophy.

On March 14, 1958, an exploratory operation was performed, through a right posterolateral chest incision with the patient under hypothermic anesthesia of 28° C. The lungs were found to be firm and fibrotic, and the heart generally enlarged with a giant left atrium. Temporary occlusion tapes were placed around the superior and inferior venae cavae, with a Satinsky clamp across the ascending aorta. Two stay sutures were placed into the left atrium. Both venae cavae were occluded, followed by occlusion of the aorta 30 seconds later. The myocardium was protected during the occlusion period by coronary perfusion with arterialized blood, as described elsewhere.[20] A large myxomatous mass was discovered filling most of the left atrium and extending through the mitral valve orifice into the left ventricle. It was extracted with some difficulty, due to the fact that it extended into the ventricle. The mass was attached to the posterolateral wall of the atrium, remote from the fossa ovalis. The base, which measured about 2 cm. in diameter, was cut off with scissors. The mitral valve was then inspected. The annulus was greatly dilated, but the chordae tendineae appeared normal and there was no concomitant stenosis. However, the valve edges appeared somewhat thickened. Two mattress sutures were placed over Ivalon pledgets across the posterior commissure with immediate disappearance of the regurgitant jet. The atrium was allowed to fill with blood and then was closed with a continuous suture of 2-0 silk. The chest was then closed in a routine manner and the

patient was rewarmed. His postoperative course was completely uneventful. The patient was not confined to bed after the second day and has remained free of symptoms since (Fig. 3).

Surgical specimen: The specimen consisted of two oval, dark red-tan shiny masses, measuring 10 by 7.5 by 5 cm. and 4 by 4 by 3 cm., weighing 158 grams (Fig. 4). The surface was irregularly contoured with a somewhat polypoid layer of soft, translucent pale yellow-gray, jelly-like myxoid tissue about one centimeter thick. This material blended with firmer, red-brown, diffusely hemorrhagic, rather homogeneous tissue that exhibited rare, vaguely defined, central fibrous strands. Patches of the surface material here and there invaginated the red core.

Microscopically, the polypoid surface of the mass was covered with endothelial cells (Fig. 5). The outer zone consisted of a pale homogeneous matrix which contained scattered large endothelial cells that lay singly or in syncytial groups and occasionally formed capillaries. A few thin-walled dilated vascular channels were present. With the Gomori trichrome stain, the matrix was pale green. The van Gieson stain revealed a yellow background with scattered strands of compact delicate red fibrils, usually located somewhat below the free surface. A rich network of fine and coarse, branching or twisted reticulum fibers was demonstrated throughout the myxoid tissue. A thin layer of metachromatic material localized immediately beneath the surface was seen with the toluidin blue stain. Elastic fibers, iron, neutral fat, and periodic acid-Schiff positive material were absent, and the mucicarmine stain was negative.

More deeply, the matrix contained pools of erythrocytes with only occasional threads or patches of fibrin. A few cavernous capillaries, mononuclear cells, and fibroblasts were present. In a minority of fields, disoriented, highly atypical, large arteries were set in hyalinized collagen (Fig. 6). These vessels had thick media with hypertrophic longitudinal or disoriented smooth muscle fibers, and fibrous intima obstructed the lumen. Rare elastic fibers were confined to the adventitia and media, and internal elastic membranes were not observed. In some places, the fibers of the media and adventitia were heavily impregnated with calcium, and the Prussian blue stain revealed abundant iron pigment (Fig. 7). Some thin-walled cavernous sinuses were bordered by strands of endothelial cells which tended to form capillaries.

The gross and histologic features of this lesion were those which have been described repeatedly as characteristic of cardiac myxomas. The gelatinous material proved to be young connective tissue rich in reticulum fibers and devoid of elastin. Mucin was not demonstrated. The stroma was sparsely cellular but was invaded from the surface by endothelium. Centrally, mature collagen, several tangles of arteries with atypical structure, and iron and calcium incrustation were present. Lamination, characteristic thrombus-organization, and pigmentation with iron, which typify mural thrombi, were absent.

Comment.—Whether such polypoid intracardiac masses are tumors or thrombi has been debated since their discovery. Ewing[16] concluded that the majority of cardiac myxomas were probably genuine and referred to the theory of their origin from superfluous embryonal tissue near the foramen ovale. Prichard,[36] who provided the most comprehensive recent review of the literature, considered them all to be tumors. Most of the authors of subsequent case reports have accepted the theory of neoplastic origin.[3, 7, 8, 18, 19, 23, 26, 27, 35, 36, 46] Myxomas have characteristic atrial locations, are attached by a stalk, do not contract, and lack stratification. Transitional forms between myxomas and thrombi have not been described.

Saphir[43] advocated a more conservative interpretation. He strongly questioned the occurrence of true cardiac myxomas mainly on the basis of the difficulty in differentiating them from the end-stages of organized thrombi.

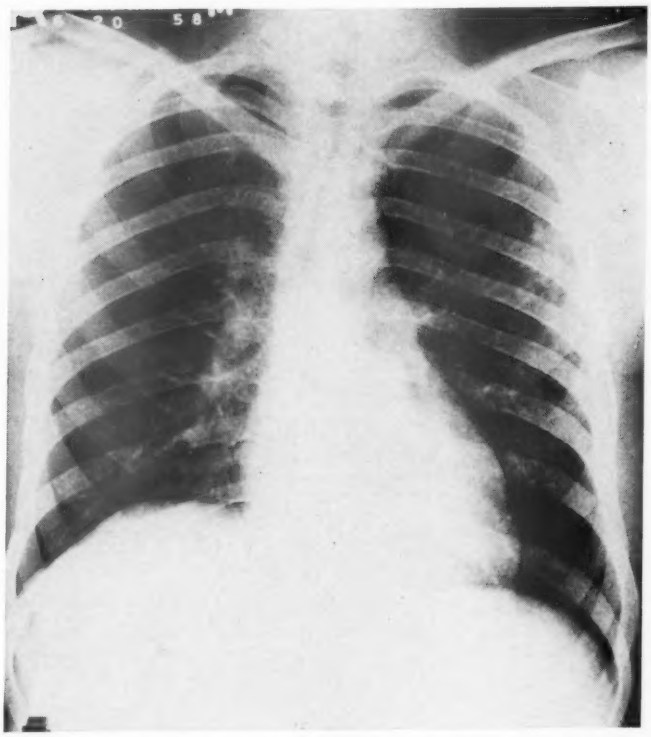

Fig. 3.—Postoperative chest film showing return of the heart size to normal.

Fig. 4.—Gross appearance of the endocardial mass.

Fig. 5.

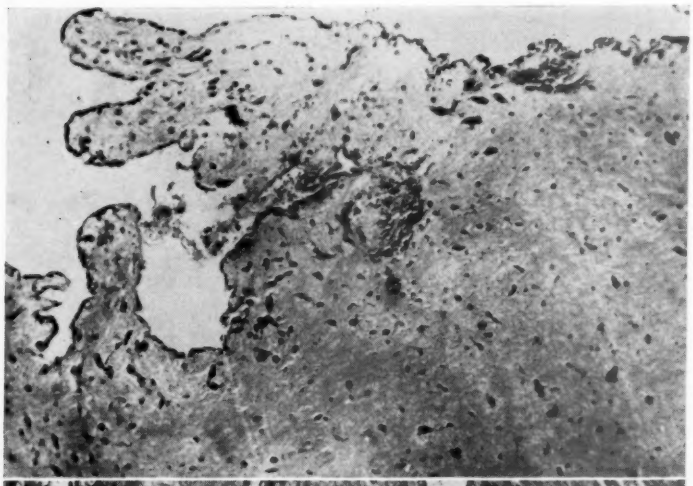

Fig. 6.

Fig. 7.

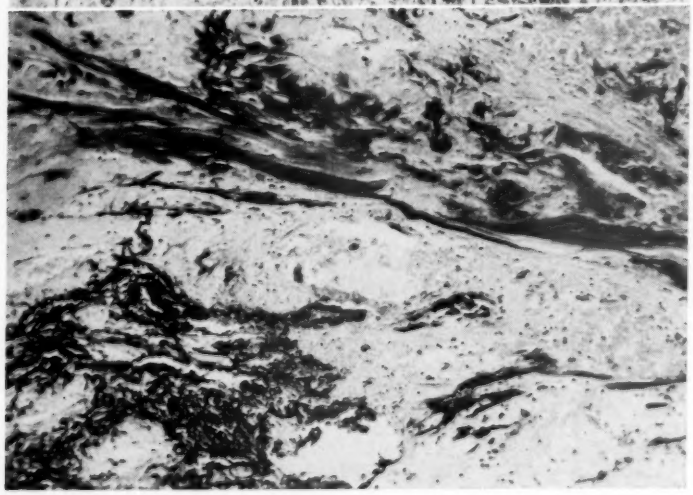

Fig. 5.—Polypoid surface layer. (Hematoxylin and eosin, × 210; reduced ⅕.)

Fig. 6.—Large blood vessels with irregularly distributed smooth muscle fibers. (Gomori trichrome stain, × 90; reduced ⅕.)

Fig. 7.—Calcification of fibers. (Hematoxylin and eosin, × 90; reduced ⅕.)

He was skeptical that definite proof of neoplasia would be forthcoming. Likoff and his associates[29] appeared to favor this view. They stated that the tumor in their case resembled a well-organized thrombus; the patient also had symptoms and pathologic changes compatible with chronic rheumatic endocarditis.

Evidence of neoplasia was unconvincing in our case. The appearance of the stroma and the number and type of the blood vessels were indicative of a reactive process. The arteries were probably located in the oldest part of the lesion and formed a minor part of its bulk. In no way did their presence suggest a hamartoma or angioma.

Whether neoplastic or thrombotic in origin, the pathogenesis of "myxomas" has not been fully clarified. They are not complications of valvular or myocardial disease. The possibility that they are initiated as thrombi, overlying a patch of inflamed endocardium, has been suggested by the febrile episodes which were noted in the course of the illness of this patient as well as other patients. However, there is no strong evidence to support such an origin.

The theory that small atrial mural hamartomatous rests are the source of these growths remains the most reasonable explanation of their origin. Prichard[36] observed endothelial spaces in the atrial wall near the fossa ovalis; the presence of most of the heart's connective tissue here and occasional endocardial deformities accounted for the location of the tumors. Shortly afterward, Raeburn[38] stated that myxomas "arise from elements of pre-existing intramural hamartomas" and that they should be termed "hamartoblastomas." For convenience they might better be described as angiomyxomas. As the myxoid features probably were due in part to degeneration of the connective tissue, the term myxoma was undesirable. Protheroe[37] recently accepted this concept of the hamartomatous origin. Anderson and Dmytryk[1] considered that the epithelium-like cells, forming gland-like spaces, which were present in their tumor possibly represented the inclusions of pericardial elements in the atrial wall during cardiac development. The myxomatous and angiomatous features were believed to be the result of changes in associated thrombotic material.

A most interesting interpretation was advanced by Ringertz,[40] who observed limited growth of cerebral and abdominal emboli from a cardiac myxoma. He believed that these masses were neither tumors nor thrombi but were proliferations of endocardial mesenchymal cells, which derived nourishment from the blood plasma. He compared this situation to a tissue culture in vivo. When the emboli became surrounded by connective tissue and were deprived of their blood supply, they ceased to grow.

We regard the lesion as a peculiar form of reaction and thrombosis which is probably based on a hamartomatous endocardial rest. With organization of the clot, enlargement could proceed through the imbibition of fluids from the circulating blood and from internal hemorrhage. In any event, the final result is a mass which has the clinical and gross appearance of a tumor that is susceptible to surgical excision.

DISCUSSION

When Prichard[36] reviewed 127 primary myxomas of the heart reported in the literature, not one had been diagnosed during the patient's life. However, Mahaim's[31] prediction, in 1945, that one day an accurate intra vitam diagnosis would be possible, soon became true. Twenty-one cases have been reported since 1951 in which the diagnosis was made ante mortem. With the advent of open heart surgery, many attempts at extirpation of a recognized myxoma have been made, as had been suggested already by Coulter[10] in 1950.

In 5 patients, the diagnosis was made during life, but no attempt at surgical removal was made or the patient died before any operation could be undertaken.[5, 23, 26, 35, 46] In 11 patients, excision of the tumor was attempted and failed, the patient dying during or immediately following surgery.[2, 4, 9, 19, 28, 41, 46, 48] In one,[34] the tumor was only partially removed due to a technical failure.

It is very interesting, and perhaps significant, that in almost all cases mentioned in the two preceding groups the correct diagnosis was not made preoperatively but rather was made during an attempted mitral commissurotomy.

The first successful operative removal of a myxoma from the left atrium was made by Craaford[11] in 1954. Seven more have since been excised by Scannell,[44] Bigelow,[6] Bahnson,[3] Robertson,[42] DeBakey,[14] Lillehei,[30] and Gerbode.[17]

A correct diagnosis of a left atrial myxoma should be possible in life for, even though it may closely resemble mitral stenosis in its clinical picture, there are certain distinguishing features. The "classical" clinical features, as best described by Protheroe,[37] Paquet,[35] and Harvey,[22] can be summarized briefly as:

1. Sudden onset of signs and symptoms.
2. Symptoms intermittent in nature, often varying with body position.
3. Palpitation, pain, giddiness, dyspnea, hemoptysis out of proportion to the degree of demonstrable heart disease.
4. Frequent attacks of Stokes-Adams syncope, usually brought on by change in body position.
5. Preference for certain body positions.
6. Intermittent febrile episodes and petechiae.
7. Relentlessly progressive right heart failure not responding to digitalis and diuretics.
8. X-ray and auscultatory findings of typical mitral stenosis, sometimes associated with mitral insufficiency.
9. Changing cardiac murmurs with change in body position.
10. Changing cardiac murmurs on repeated examinations over a period of time.
11. Low blood pressure and pulse pressure, frequently associated with cyanosis of the extremities.
12. Terminally, intractable pulmonary edema and peripheral circulatory collapse.

These clinical features can, of course, be logically explained on the basis of the pathologic changes present in this condition, namely: (1) obstruction of left atrial flow, (2) left atrial enlargement, (3) decreased left ventricular output, (4) increased pulmonary volume and pressure, and (5) right ventricular enlargement.

On the basis of these pathophysiologic changes it becomes understandable why the picture of mitral stenosis is so closely mimicked. Auscultation, x-ray, fluoroscopy, and the electrocardiogram are of little or no help. An opening snap has been reported in 8 cases,[28, 45] and even the phonocardiogram has been shown to be characteristic of mitral stenosis.[23, 28, 29] Right heart catheterization cannot add anything of diagnostic value and appears only to confirm the clinical diagnosis of mitral stenosis. To the knowledge of the authors, Craaford's case and the case herein reported are the only 2 cases where combined right and left cardiac catheterization was carried out. However, this procedure, too, added no clue to the true nature of the condition. In the case herein reported, the findings were characteristic of pure mitral insufficiency.

It is of interest that many patients with myxoma of the left atrium do give a history of arthralgia, periods of fever and myalgia,[22] thus simulating a rheumatic fever history. In this patient, there appeared to be a chronic type of active rheumatic fever, as evidenced by the changes in the sedimentation rate and the acute phase reactants. However, there was no clear-cut history of rheumatic fever during childhood or adolescence.

Approximately 50 per cent of proved cases of rheumatic mitral stenosis do not give a history of rheumatic fever.[32] It follows that if a patient has a definite history and/or signs of rheumatic fever, these are of little aid one way or the other in the diagnosis of myxoma versus rheumatic heart disease.

Sometimes fragments of a myxoma may break off and are carried to the periphery as small emboli, simulating the picture of subacute bacterial endocarditis. However, only one case of myxoma complicated by a *Staphylococcus aureus* infection has been reported.[15]

The only possible way to arrive at an accurate ante mortem diagnosis is by left cardioangiography. Indeed, this was the case in every instance where a myxoma was correctly diagnosed preoperatively rather than having been found incidentally to an attempted mitral commissurotomy. There is no doubt that in our patient a correct preoperative diagnosis could have been made had a cardioangiogram been made. Fortunately, the surgical approach was such that excision of the tumor presented no difficulty. The severity of the valvular incompetence made it mandatory to proceed with the originally planned procedure, although one may speculate whether the mitral insufficiency would have disappeared in time without the plication.

SUMMARY

A case of successful excision of a "myxoma" of the left atrium is reported. The tumor appears to be the first one described which closely simulated pure mitral insufficiency.

The tumor was considered to be a peculiar form of reaction and thrombosis, probably based on a hamartomatous endocardial rest.

The diagnostic features of myxoma of the left atrium are briefly discussed. The only means of making a correct preoperative diagnosis is by left cardioangiography.

The combination of hypothermia and coronary perfusion appears promising and feasible in the correction of mitral insufficiency by the Lillehei modification of the Nichols operation.

ADDENDUM

Since submission of this paper for publication, we have seen a 52-year-old female patient with signs and symptoms suggestive of a left atrial myxoma simulating mitral stenosis. The diagnosis was confirmed by cardioangiography and the tumor subsequently was removed with the patient under hypothermia (J. B. G.). The patient had an uneventful recovery.

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THE IMPINGEMENT OXYGENATOR

Experimentation in the Study of a New Method of Blood Oxygenation

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MANY pump-oxygenator systems, employing various principles of blood oxygenation, are in current use. Although successful perfusion has been accomplished using oxygenators, the ideal artificial heart-lung system remains to be developed. Such an oxygenator would be small, simple, and completely safe from the standpoint of emboli and the effect on blood elements, would provide unlimited flow of adequately oxygenated blood, and would be able to be heat sterilized. It should be inexpensive to operate and maintain, require minimal donor blood, be easily cleaned, and maintain normothermia and acid-base equilibrium. The devices being used currently utilize one of three fundamental methods by which blood has been artificially oxygenated: (a) by-passing blood through a closed semipermeable membrane circuit which is surrounded by an oxygen mixture; (b) by filming blood in direct contact with an oxygen atmosphere; and (c) by bubbling oxygen through blood. Information concerning these methods is readily available.[2-11]

While searching for a more simple, efficient, and safe way of accomplishing gas exchange in blood, a method was proposed by one of us (M. R. K.). In the method proposed, a jet of oxygen impinges against a film of blood, forcing the blood into an ultrathin film, and accelerating the flow immediately beneath the jet. Consequently, a large amount of surface area per unit volume of blood was created. Spraying of blood into droplets has been shown by Björk[1] to cause excessive hemolysis, and had to be prevented.

THEORY OF BLOOD-OXYGEN INTERFACES

The following theory has been developed from the application of basic physical chemistry theory to film thickness and velocity studies of the impingement oxygenator. It applies, however, to all blood oxygenators employing a direct gas-blood interface.

It was first necessary to determine whether the blood film was in turbulent or laminar flow, since the condition of flow is fundamental to mass transfer theory. Reynold's number, R, is a dimensionless fluid dynamic value which relates the inertia and viscous forces in a moving fluid. A small r denotes that the viscous forces are predominant and that there is no mixing between

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the fluid layers. Reynold's number for the blood flow under the impinging jet, calculated from the principles of flow in an open duct at maximum flow rate, was about 120. Since a Reynold's number less than 2,000 indicates laminar flow, the blood was undoubtedly nonturbulent.

Laminar Flow Theory.—

The velocity profile of a liquid in laminar flow across a plate is shown in Fig. 1. Liquid in contact with the plate is at zero velocity, and the velocity increases with distance from the plate. The liquid is at maximum velocity at the surface and the stream may be regarded as a series of thin sheets sliding over one another. This slipping of the liquid layers results in mechanical shearing forces, which, incidentally, may have a destructive effect upon the suspended formed elements of the blood.

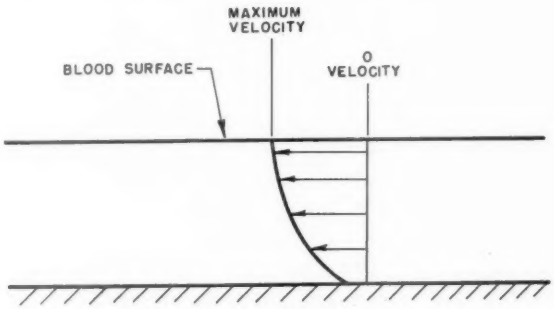


Fig. 1.—Laminar flow over a plate.

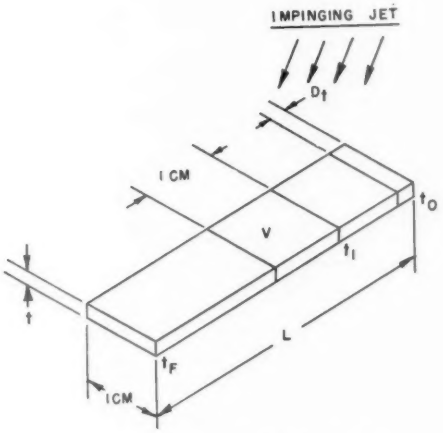


Fig. 2.—Unit blood film.

Consider the blood film flowing under the oxygen jet in the impingement oxygenator (Fig. 2). The blood contained under the surface, 1 cm. wide, is exposed to oxygen from the time, t_o, it emerges from the orifice and is forced into a thin film, to the time, t_f, at which the blood is beyond the effect of the oxygen jet. At time t_o, the film is at equilibrium at the venous oxygen concentration. During the first increment of time, dt, the transfer of oxygen into the film is extremely rapid, because the surface is at a lower concentration than the gas. By time t_1, the surface has become equilibrated with

the gas and the rate of oxygen uptake has decreased considerably. As time progresses, molecular diffusion of oxygen from the surface into the blood below the surface continues at a still slower rate. Since flow is laminar, the equilibrated blood remains at the surface, inhibiting further oxygen uptake. The blood takes oxygen only as fast as molecular diffusion can deliver oxygen to the desaturated blood below the surface.

Consider the travel of the volume of blood, v, contained under a unit surface area 1 cm. long and 1 cm. wide. Knowing the rate of oxygen uptake, N, and the time, t, during which this uptake rate is effective, the amount of oxygen absorbed is $O_2 = Nt$.

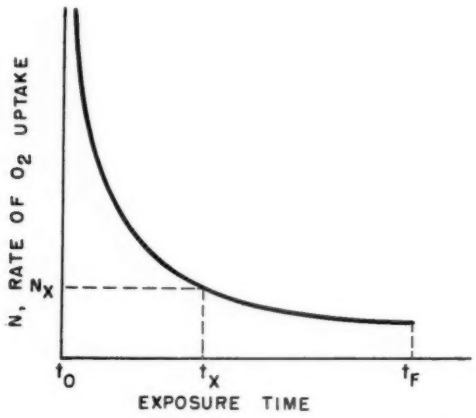


Fig. 3.—Oxygen uptake as a function of time.

However, as discussed above, the rate of oxygen uptake is constantly changing with time (Fig. 3). The amount of gas absorbed, therefore, must be stated for an infinitely small period of time.

$$dO_2 = N_x dt.$$

This means the amount of gas absorbed during the infinitely small period of time (dt) is equal to the instantaneous rate of oxygen uptake, N_x, at the particular instant, t_x, multiplied by the infinitely small period of time. The summation of these small increments of absorbed gas from time t_o to t_f, during which the volume, v, traveled the entire length of film is the amount of gas absorbed, and is expressed as

$$O_2 = \int_o^f N dt \tag{1}$$

The over-all effective N during the time $t_o - t_f$ may be determined:

$$N_{ave} = \frac{O_2}{t_o - t_f} \tag{2}$$

Practical application of Equations (1) and (2): It was impractical to take blood samples at various points along the film in a device such as the impingement oxygenator. It has been common practice, however, to analyze the

oxygen content of the venous blood put into the oxygenator and the arterial blood collected after a pass under the jet. From these values, the amount of oxygen absorbed by the entire blood stream may be calculated:

$$\text{Final } O_2 \text{ content} - \text{Initial } O_2 \text{ content} = \Delta O_2 \text{ content}, \frac{\text{cm.}^3 O_2}{\text{cm.}^3 \text{ blood}}.$$

By knowing the film thickness, T, of the blood stream, the blood volume and the oxygen absorbed in this volume may be determined:

$$v = 1 \text{ cm.} \cdot 1 \text{ cm.} \cdot T \text{ cm.} = T \text{ cm.}^3$$

$$O_2 = \Delta O_2 \text{ content} \cdot v \tag{3}$$

This is the same oxygen volume expressed in Equation (1)

$$O_2 = \int_0^f N dt = \Delta O_2 \text{ content} \cdot v$$

Equation (2) becomes:

$$N_{ave} = \frac{\Delta O_2 \text{ content} \cdot v}{t_o - t_f} = \frac{\Delta O_2 \text{ content} \cdot F}{L} \tag{4}$$

Where:

F = blood flow rate in c.c./min./cm. nozzle width.
L = film length in centimeters.

It will be noted that Equation (4) is independent of film thickness. For a particular volume flow rate, the volume, v, is directly proportional to film thickness, but time is inversely proportional to T, since it is inversely proportional to velocity. The numerical value of N_{ave}, therefore, depends upon flow rate but not film thickness.

If the film length is not known, the over-all rate of oxygen uptake, U, for a unit width of film may be calculated from the increase in O_2 content per unit volume of blood and the flow rate:

$$U = \frac{\Delta O_2 \text{ content}}{\text{cm.}^3 \text{ blood}}, \frac{\text{cm.}^3 \text{ blood}}{\text{min./cm. nozzle}}$$

$$U = \Delta O_2 \text{ content} \times F, \frac{\text{cm.}^3 O_2}{\text{min./cm. nozzle}}. \tag{5}$$

Equation (5) is the common calculation for oxygenator studies.

It was stated in the basic physical chemistry theory that the rate of oxygen uptake per unit surface area is a function of both the mass transfer coefficient and the driving force. The driving force at any point in the stream is equal to the difference in oxygen concentration in the gas and the concentration existing at the point under study. The partial pressure of oxygen in 35 per cent saturated blood is about 20 mm. Hg and at 97 per cent saturation the pressure is about 100 mm. Hg. The driving force between pure oxygen gas at one atmosphere and the blood at 35 per cent saturation is

760 mm. Hg – 20 mm. Hg = 740 mm. Hg ppO_2.

The force between O_2 gas and 97 per cent saturated blood is

760 mm. Hg – 100 mm. Hg = 660 mm. Hg ppO_2.

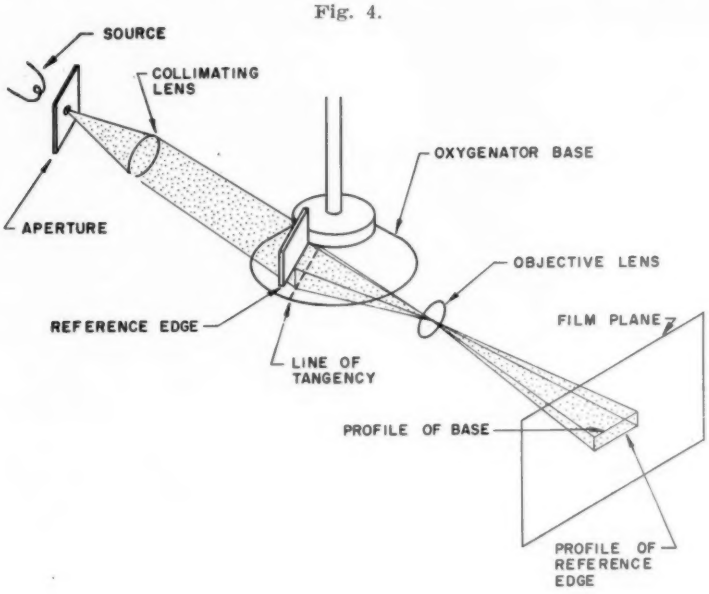
The average driving force in a volume of blood being raised between these limits of saturation is 700 mm. Hg ppO_2. If the driving force is considered to be constant at 700 mm. Hg, the maximum error would be 6 per

cent. This does not represent a significant error, and, for the simplicity of calculations, the driving force has been considered constant.

Film Thickness and Velocity.—

To study the interrelationships of blood film thickness, blood velocity, oxygen uptake rate, and jet pressure, a profile photomicrograph was con-

Fig. 4.



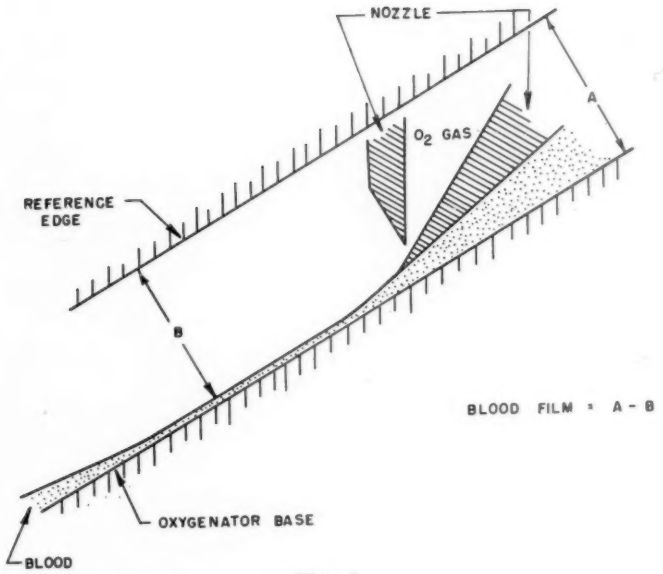


Fig. 5.

Fig. 4.—Profile photomicrograph.
Fig. 5.—Blood film measurement.

structed. The instrument consisted of a coherent light source, the optical axis of which was interrupted by the conical base of the oxygenator. The profile of the base was imaged on the film plane of a camera (Fig. 4). In one

frame the distance from the profile of the dry base to a reference edge was measured. This was compared to the distance from the same reference to the blood film profile in a subsequent frame (Fig. 5), the difference being the thickness of the blood film. The instrument allowed measurement of film thickness to an accuracy of about 5 μ.

Film velocity, V, equals F/T. The calculations showed that, in general, both film thickness and velocity varied directly with blood flow rate, indicating that as flow rate increased film thickness increased, but not in proportion to the increase in flow. It was shown also that film thickness was inversely proportional to jet nozzle pressure. For some unexplained reason film thickness and velocity were approximately equal with 0.002 and 0.006 inch jet openings, thicker, slower films occurring with a 0.004 inch nozzle opening. Fig. 6 shows the results at 0.002 and 0.004 inch jet openings.

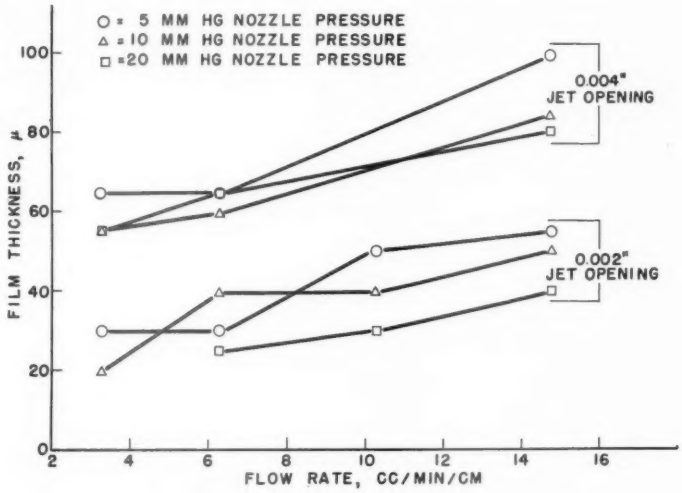


Fig. 6.—Results of blood film experiments.

Seven samples of citrated bovine blood were run through the oxygenator on Feb. 4, 1957. In addition to profile photomicrographs of the blood film, Van Slyke oxygen measurements were taken from the samples. The results are shown in Table I. With the exception of one value (which resulted from the highest nozzle pressure used), oxygen uptake rate varied directly with

TABLE I. DATA ARE FOR BLOOD FILM 1 CM. WIDE
(0.004 IN. BLOOD OPENING—0.004 IN. GAS JET OPENING)

SAMPLE NO. (MONTH, DATE, TEST NO.)	NOZZLE PRESSURE (CM. Hg)	FLOW RATE (CM.^{3}/MIN./CM.)	MIN. FILM THICKNESS (μ)	ΔO_2 CONTENT (CM.^{3} O_2/CM.^{3} BLOOD)	$U = \Delta O_2$ CONTENT · F	FILM VELOCITY (CM./MIN.)
2.4.2	5	3.48	80	0.0414	0.144	435
2.4.3	10	3.48	65	0.0498	0.178	535
2.4.4	10	6.50	85	0.0390	0.253	765
2.4.5	10	10.65	100	0.0242	0.258	1065
2.4.6	10	15.70	95	0.0231	0.363	1655
2.4.7	5	6.50	95	0.0325	0.211	685
2.4.8	20	6.50	75	0.0603	0.392	867

film velocity (Fig. 7). This means that, assuming the film lengths to be essentially equal, the *rate* of gas transfer decreased as exposure time increased. As suggested by the above theory, it is, therefore, efficient to present as large a blood surface per unit time as possible to oxygen gas with a short exposure time. Oxygenation rate will increase, for example, if, in a given apparatus, the surface area per unit time is doubled by reducing exposure time by a half.

This concept is contrary to the ideas of Melrose[10] and Björk,[1] who state that there is an optimum exposure time beyond which oxygen uptake rate decreases. The theory presented here states that oxygen uptake rate is *constantly* decreasing from the *instant* of exposure, and *there is no finite minimum exposure time* beyond which oxygenation decreases.

It is inefficient to attempt to saturate the entire volume of blood contained in a film in a single exposure—when a film contacts oxygen, the surface plasma becomes rapidly saturated, but further oxygenation takes place only as fast as molecular diffusion can deliver oxygen molecules to blood below the surface. Consequently, it is desirable to move the saturated surface into the blood mass *as fast as possible* and bring unsaturated plasma to the surface. The saturated plasma which has been brought into the blood mass will then deliver oxygen to the red cells.

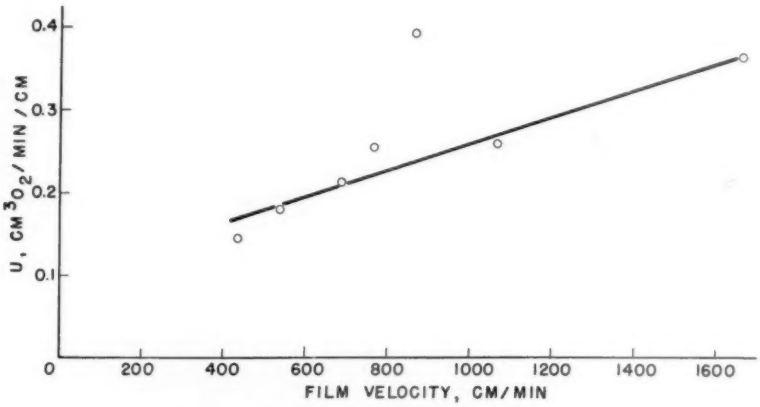


Fig. 7.—Film velocity vs. oxygen uptake.

In applying the theory of mass transfer to a thin laminar stream of blood, two assumptions have been made which must be noted. The suspension of red cells in plasma has been regarded as a homogenous solution with oxygen being absorbed in solution rather than in chemical combination. The amount of gas held in solution by a liquid is directly proportional to the partial pressure of the gas in the liquid. It will be noted from the oxyhemoglobin dissociation curve of blood, however, that as the pO_2 increases beyond 60 mm. Hg, the quantity of O_2 contained in the blood increases only slightly. The blood thus becomes saturated with the gas, a phenomenon which would not occur for a true solution of a gas in a liquid. Between 0 to 60 mm. Hg pO_2, the dissociation curve is sufficiently straight for the calculations in this report. This corresponds to saturation of 0 to 90 per cent. Saturation values above 90 per cent have not been used in the O_2 uptake coefficient calculations.

The suspension of red cells in the plasma adds considerable complexity to the physical analysis of the system. If the diffusion of oxygen molecules through the red cell membrane and the chemical combination of oxygen with hemoglobin were controlling the process (that is, if the plasma were delivering oxygen to the cells at a faster rate than the cells could absorb it), the rate of oxygen uptake would be constant until saturation was reached, and there would be no concentration gradient throughout the film depth. This, however, has not been found to be true experimentally, and it appears that the red cells absorb oxygen as fast as it is delivered to the cells by the surrounding plasma. Consequently, the foregoing analysis has been based on the apparently sound assumption that the rate of solution and diffusion of oxygen in the plasma controls the process.

DESCRIPTION OF OXYGENATOR

In the device shown in Fig. 8, gaseous oxygen emerged from the narrow nozzle orifice and impinged on the oxygenator base. The gas, changing direction as it reflected off the base, imparted a certain amount of its kinetic energy to the blood flowing over the base. This energy was consumed as work used to overcome the viscosity and surface tension of the blood, thus forcing the liquid into a film and accelerating it.

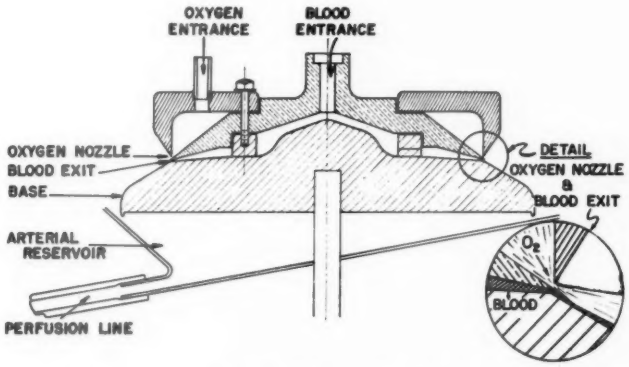


Fig. 8.—Model E impingement oxygenator.

Since pressure is everywhere equal in a fluid, the impinging jet created a "back" pressure as well as a "forward" pressure. Consequently, the blood had to be forced under the jet by positive pressure through a closed entrance system. It was practical, therefore, that the oxygenator consist of a single nozzle, since a pump or considerable gravity head would be required for each stage of a multiple-stage oxygenator.

Earlier models of the device led to the eventual design of the knife-edge lip configuration shown in Fig. 8. In a previous design, the oxygen jet was released a short distance above the blood orifice. Due to the high gas velocity, a reduced pressure formed between the jet and the oxygenator base. This reduced pressure drew blood up toward the oxygen exit where it was picked up by the overpassing jet and formed into a fine spray. The knife-edge lip configuration avoided the reduced pressure area and eliminated the blood spraying problem.

The final oxygenator unit (Model E) as shown assembled in Fig. 9, and disassembled in Fig. 10, was of solid steel construction, weighing 39 pounds, and composed of three basic precision lathed parts, the top, middle, and base sections. The base measured 25.5 cm. in its largest diameter, and the circumference of the nozzle was 60.0 cm. These parts were bolted together, utilizing brass shim stock rings between parts to adjust the oxygen and blood nozzle openings. When assembled, two completely separate chambers were created, one distributing oxygen and the other blood in a uniform manner to the respective nozzle openings. The single opening in the top of the middle section

Fig. 9.

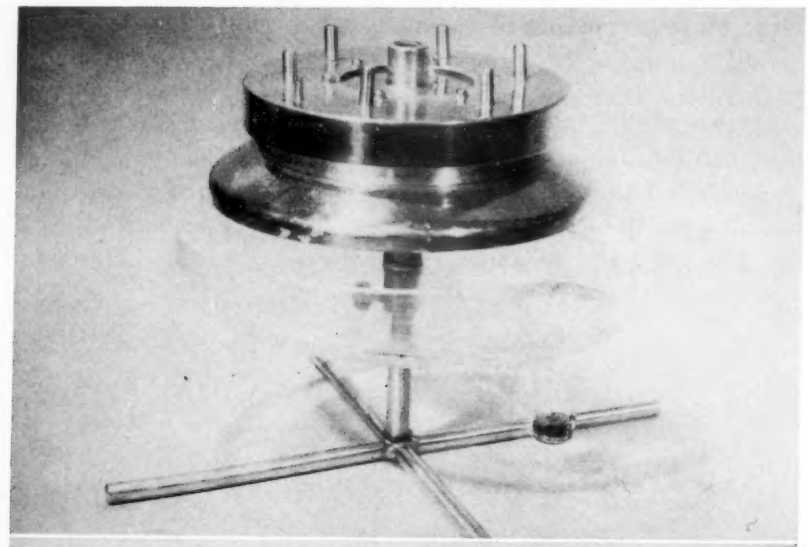


Fig. 10.

Fig. 9.—Oxygenator assembled.
Fig. 10.—Oxygenator disassembled.

received blood from the small Lucite reservoir, and oxygen was supplied through the multiple ports on the top section. A smaller port permitted measurement of oxygen nozzle pressure. The three main sections were gold-plated, since previous studies showed this metal to be most resistant to corrosion by blood. The oxygenator rested on a stainless steel frame which also supported a Lucite arterial reservoir.

Due to the tremendous oxygen flow rates required, a large drum-type gas humidifier was constructed. From this humidifier, oxygen was distributed by a manifold to the oxygenator through rubber tubes.

IN VITRO BLOOD TESTS

Extensive dog and bovine blood tests were performed to determine optimum operating conditions. It was found that, in general, oxygen uptake increased with jet pressure and blood flow rate. It was desirable to enter the blood close to the jet (where the gas velocity was high). The minimum width of the blood orifice was limited, however, by permissible blood pressure drop across the orifice; 0.004 inch (0.102 mm.) was found to be satisfactory.

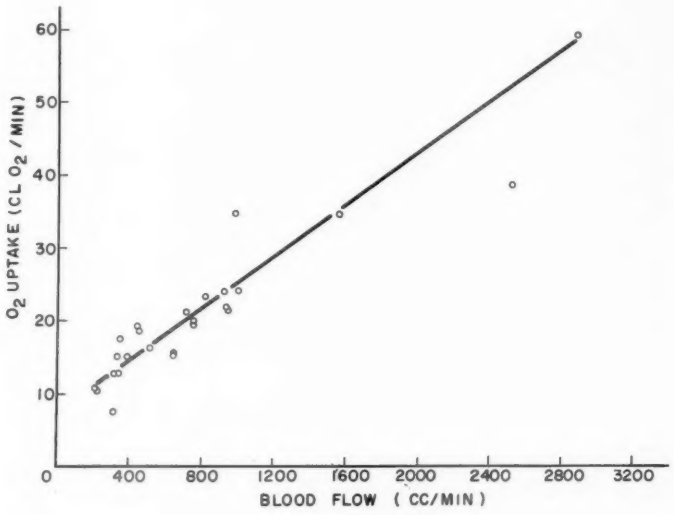


Fig. 11.—Flow rate vs. oxygen uptake.

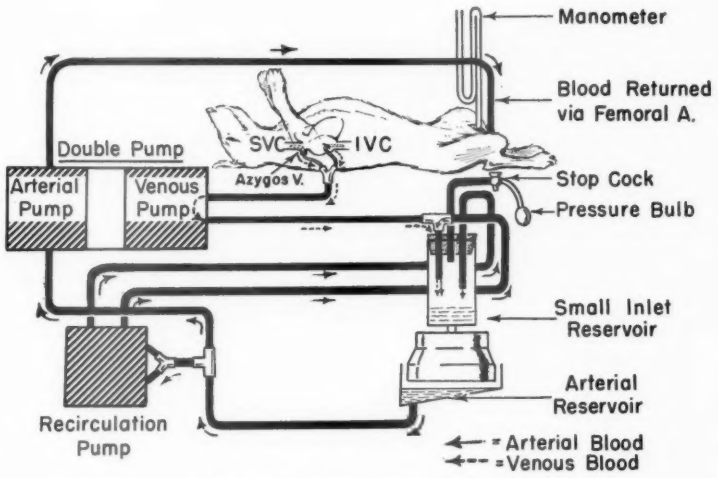


Fig. 12.—Perfusion circuit.

The consumption of gaseous oxygen was impractically high, the jet opening and pressure being somewhat limited by the length of operating time which could be obtained from two 6,900 L. oxygen bottles. The optimum oxygen nozzle opening was found to be 0.004 inch and the highest practical nozzle pressure was 10 cm. Hg. Oxygen uptake rate versus blood flow rate

TABLE II

EXPER. NO.	LENGTH (MIN.)	WT. (KG.)	PERFUSION RATE (C.C./MIN.)	PERFUSION RATE (C.C./KG./MIN.)	RECIRCULATION RATE (C.C./MIN.)	PER CENT O_2 SATURATION		O_2 CONTENT		O_2 CONSUMPTION (C.C./MIN.)	pH (ART.)	CO_2 CONTENT (ARTERIAL) (VOL. %)	PCO_2 (ARTERIAL) (MM./HG)	PERFUSION MEAN ART. BP (MM./HG)	PLASMA HEMOGLOBIN INCREASE (MG. %)	TEMPERATURE RANGE (DEGREES C.)
						ART.	VEN.	ART.	VEN.							
						(1) 86.0	60.0	18.85	12.82		(1) 7.41	34.0	28.5			
						(2) 68.0	53.0	14.92	11.31			40.0	41.7			
62	14.5	10.0	350	35.0	1,500	(3) 64.0	28.0	13.83	6.50	25.6	(3) 7.33	41.0	42.0	45-72	38.0	—
						(1) 121.0	43.0	20.95	7.54		(1) 7.48	27.76	20.0			
						(2) 91.0	50.0	15.67	8.80			34.9	46.0			
114	14.1	9.0	350	38.0	1,500	(3) 87.5	48.0	15.40	8.45	24.3	(3) 7.21	36.54	46.0	25	53.0	35-32
						(1) 103.0	69.0	20.88	13.96		(1) 7.35	24.0	23.5			
						(2) 78.0	39.0	15.74	7.95			35.0	33.5			
3	13.5	9.0	350	38.0	1,150	(3) 59.0	25.0	12.01	5.09	24.2	(3) 7.29	36.0	40.0	30-45	30.0	37-30
						(1) 69.0	35.6	11.85	6.12		(1) 7.10	13.0	20.0			
						(2) 46.0	10.4	7.91	1.79			23.0	36.0			
57	16.3	10.0	350-500	34-50	1,985	(3) 55.0	14.8	9.45	2.54	34.6	(3) 6.95	24.0	50.0	20-45	34.0	36.5-28.0
						(1) 106.0	50.0	20.30	9.64		(1) 7.20	21.0	26.5			
						(2) 101.0	45.0	19.32	8.71			28.0	47.5			
109	21.2	10.5	350	32.7	3,480	(3) 94.5	39.0	18.05	7.56	36.8	(3) 7.01	30.0	58.7	50-70	201.0	37-30
						(1) 106.0	19.9	14.10	2.65		(1) 7.37	20.5	17.3			
						(2) 65.0	12.5	8.65	1.67			25.6	36.0			
145	24.0	7.9	343	43.5	3,480	(3) 102.5	12.6	14.89	1.68	45.3	(3) 7.10	23.23	34.5	12-24	106.0	36-32.8

						(1) 109.0	63.5	13.88	7.75		(1) 7.30	28.9	27.0					
						(2) 102.0	74.6	13.16	9.49			35.5	46.0					
67	18.5	9.0	390	43.5	3,480	(3) 92.3	43.2	13.46	6.32	27.8	(3) 7.15	35.7	48.0	75-90	106.0	38-33		
						(1) 77.9	53.7	10.72	7.25		(1) 7.34	24.5	22.8					
						(2) 98.5	75.0	16.23	12.40			27.2	27.3					
135	17.5	10.7	571-698	65.2-53.3	3,480	(3) 88.5	57.4	14.59	9.46	29.2	(3) 7.16	26.6	39.0	104-184	209.0	37-31		
						(1) 92.0	46.2	15.01	7.59		(1) 7.30	26.2	27.0					
						(2) 55.7	21.0	10.01	3.80			35.6	49.5					
61	21.3	10.5	632-724	60.0-69.0	3,480	(3) 54.4	22.2	10.32	4.22	38.6	(3) 7.07	34.5	56.0	46-60	151.5	37-35		
						(1) 85.4	47.6	13.26	7.40		(1) 7.19	28.1	35.4					
						(2) 114.0	67.0	18.96	11.10			28.2	31.2					
108	20.8	9.0	500-275	55.5-30.6	3,480	(3) 112.0	78.0	19.30	13.27	16.6	(3) 7.08	26.1	29.6	65-115	218.0	37-33		
						(1) 91.0	62.8	18.29	12.63		(1) 7.19	34.2	45.0					
						(2) 76.5	45.6	15.57	9.39			37.7	66.0					
140	20.3	10.7	522-625	48.0-58.0	4,045	(3) 85.0	53.9	17.08	10.82	32.6	(3) 7.04	35.4	64.0	90	448.0	38-32		
						(1) 105.0	18.0	15.60	2.66		(1) 7.36	26.75	23.8					
						(2) 79.2	60.0	13.85	10.50			28.5	35.0					
11	31.5	12.55	400-600	32.0-48.0	3,480	(3) 111.0	71.0	16.85	10.72	24.5	(3) 7.02	28.4	52.0	55-190	149.0	36.5-30.5		
						(1) 109.0	52.8	16.88	8.04		(1) 7.42	26.8	21.9					
						(2) 55.5	30.2	10.41	4.77			31.1	46.5					
71M	25.9	14.4	540	32.5	3,480	(3) 36.7	8.4	6.9	1.57	28.8	(3) 6.93	31.0	60.0	90-120	122.0	38-34		

at these operating conditions is plotted in Fig. 11. The tendency toward direct variation of oxygen uptake with flow rate indicated the desirability of obtaining as high a blood flow through the nozzle as practical.

To realize the elevated oxygen uptake rate at high flow rates, the oxygenator was incorporated in a multiple pass recirculation system (Fig. 12). By dividing the recirculation flow through two latex tubes in a 6S Sigmamotor pump, recirculation rates as high as 4,045 c.c. per minute were obtained. It was anticipated that this would result in an oxygenation rate greater than 60 c.c. O_2 per minute.

The data obtained from dog perfusions using this apparatus are tabulated in Table II. In these relatively short perfusions with the impingement oxygenator, severe physiologic deficiencies have been noted, the most important being its limited capacity to oxygenate blood adequately. Carbon dioxide was not effectively eliminated from the blood by the oxygenator, with resulting respiratory acidosis. Excessive destruction of red cells by the oxygen jet occurred in even short perfusions with this device. These and other mechanical disadvantages of the impingement oxygenator would prohibit its use clinically.

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EXPERIMENTAL CORONARY ARTERY SURGERY IN DOGS EMPLOYING A PUMP-OXYGENATOR

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IN THIS country the number of persons who die annually of coronary ischemia exceeds half a million.[4] Examination of some of these hearts reveals that well over 80 per cent have adequate cardiac muscle but that the organ failed as a pump, either because a narrowed coronary artery became acutely occluded or could not, under stress, carry a sufficient amount of blood.[17, 18, 25] A considerable number of hearts show no muscle degeneration whatever.[5]

For many years this problem was considered entirely medical and treatment, except for the use of digitalis, was directed largely toward lightening the work load of the heart.

There are now available a number of methods designed to increase the blood flow to an ischemic or potentially ischemic myocardium (Table I). Some of these are still in the experimental state; some have found a much-disputed degree of clinical acceptance.[4, 9, 23] None are applicable to the whole group of patients with coronary disease.

Some of these direct techniques are already in use. Patients in acute coronary failure have been supported on pump-oxygenators[19] and coronary angiography performed safely in those with advanced coronary disease.[22] Operative attacks have been made on the coronary arteries themselves.[1, 2]

TABLE I. SURGICAL METHODS DESIGNED TO INCREASE THE BLOOD SUPPLY OF CARDIAC MUSCLE

1. Creation of adhesions between myocardium and pericardium by destroying pericardial space—mechanically (Beck I) or chemically[4, 11]
2. Creation of adhesions between myocardium and adjacent tissues (pericardium, lung, muscle, spleen, intestine)
3. Increasing venous blood pressure in coronary sinus[3, 11] by:
   a. Narrowing or ligating sinus
   b. Anastomoses of artery to sinus
   c. Both
4. Increased blood flow through pericardiophrenic system by ligation of internal mammary artery[9]
5. Increased coronary flow resulting from pericoronary neuronectomy or dorsal sympathectomy
6. Localized increase in myocardial blood supply by implantation of internal mammary artery[23]
7. Coronary endarterectomy[1, 2]
8. Direct anastomoses between the coronary arteries and the systemic circulation[21]

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It is quite possible that in the near future the patient with coronary artery disease, either acute or potential, may be taken to a cardiac center, supported on a pump-oxygenator while his coronary and myocardial condition is evaluated by electric, radiographic, and chemical methods, and that one or a combination of surgical procedures will be employed to correct or modify his myocardial circulation.

The purpose of this report is to present our experience with coronary artery surgery performed in the dog. In addition, coronary angiography was studied and three different oxygenators were evaluated for later use in human beings.

A total of 50 dogs was employed. All were subjected to perfusion and coronary artery surgery. Three machines were used. The first was a bubble oxygenator* of disposable type modeled after that described by Lillehei and his group.[8] The second was a filming oxygenator† developed by Melrose[15] of England, and the third was a modification of the disc oxygenator originally described by Björk.[6] Dog blood for priming the oxygenator was obtained by exsanguinating mongrel dogs that had received succinylcholine chloride (Anectine) while on a respirator. If the blood was to be used the same day, no antibiotic was added; however, if the blood was to be stored overnight in the refrigerator, a small amount of Terramycin or penicillin and streptomycin solution was added.

METHOD

With morphine as a preanesthetic agent we first employed Nembutal for basal anesthesia but, finding that many dogs were unduly sedated, we changed to Pentothal which was more satisfactory. The dog was placed on the table in a right posterolateral position protected from an underlying heating pad with paper insulation and was intubated with a tracheal cannula. Unless the respirations were considerably depressed the animal was not placed on a respirator until just before thoracotomy.

After the peripheral vessels had been exposed surgically, a small cannula for the delivery of fluid or medicine was placed in the femoral vein on one side, and on the opposite side a plastic catheter was inserted until its tip lay in the inferior vena cava. A similar cannula was introduced through the left jugular vein into the superior vena cava and the arterial cannula into the right carotid. Prior to the insertion of the cannulae, the animal was given 1 mg. heparin per kilogram of body weight. A second milligram per kilogram was given intravenously just before perfusion.

The left side of the chest was opened through the fourth intercostal space. The azygos vein was ligated and tapes for later occlusion were passed about the two cavae and about the ascending aorta proximal to the brachiocephalic trunk. The pericardium was opened. An area of either the left anterior descending or the left circumflex coronary artery as free of side branches as possible was

*Manufactured by Travenol Laboratories, Morton Grove, Illinois.
†Built for us by Mr. L. Hercik of the Hill Acme Company, Cleveland, Ohio.

dissected free of epicardium and fat. While this was being done, other members of the team primed the sterilized oxygenator with blood warmed to body temperature.

The oxygenator was started and once the flow was satisfactory the two caval tapes were tightened, the aorta occluded, and the heartbeat stopped by injection into the ascending aorta just below the occluding tape a solution of 2 c.c. of 25 per cent potassium citrate in 18 c.c. of blood. The caval tapes were then released to prevent overdistention of the heart.

Next, the coronary artery which had been designated for surgery was divided completely and a stent of plastic or copper wire introduced into the divided vessel. The vessel was then reanastomosed, end-to-end, with interrupted sutures of No. 6-0 silk on an atraumatic needle. Once the anastomosis was complete the stent was removed either through a small hole in the distal coronary branch which was repaired or it was slipped backward into the ascending aorta and removed through a small aortic incision which was then closed. In three instances the stent was introduced through the aorta itself but considerable time was wasted in locating the coronary ostium and this method was given up.

When the aorta was released, the heart either started spontaneously or it fibrillated and was returned to normal sinus rhythm by electroshock or by a second potassium arrest. During chest closure 150 mg. protamine sulfate was given slowly intravenously in 200 c.c. of 5 per cent glucose in distilled water.

The dogs were divided into groups according to the type of oxygenator employed. Tables II and III outline the procedures and results. In Groups 1 and 2 a simple division and reanastomosis was performed as described. In Groups 3 and 4 an anastomosis was made between a branch of the left subclavian (usually the internal mammary) and the circumflex or anterior descending coronary artery. After arrest the coronary artery to be employed was ligated proximally and divided. In Group 3, because of aortic bleeding from the needle puncture the cardiac arrest was accomplished by injecting the potassium citrate solution through a catheter (No. 7 F.) introduced via the brachial artery to a point just above the aortic valve. The catheter was withdrawn beyond the occluding aortic tape after the heart had stopped. The same catheter in Groups 4 and 5 was introduced through a subclavian branch and used as a stent for the anastomosis after being withdrawn from the aorta. Because of cerebral damage in the third group of dogs the carotid artery was no longer employed for perfusion and the arterial blood from the pump-oxygenator was introduced into the femoral vessels in Groups 4 and 5.

The fifth group was composed of animals in which the subclavian artery or a branch thereof was anastomosed to the coronary artery through an interposed freeze-dried graft* prepared from mongrel dogs. In 5 animals in this group a bulldog clamp was placed across the base of the sinus of Valsalva, the sinus wall was divided, and the aorta was closed with a running silk suture. Thus the anterior descending, circumflex, and septal branches were perfused via the left subclavian artery.

*These grafts were prepared for us by Mr. Frederick Olmstead and we are grateful for his help.

In certain animals in the third, fourth, and fifth groups, angiograms of the coronary arteries were made at the time of initial arrest and repeated after the anastomoses had been performed. This technique and its results has been reported in a previous paper.[16]

RESULTS

The operative deaths occurred in those dogs which were unable to support an adequate blood pressure once they were removed from the pump-oxygenator, or dogs in which bleeding was uncontrollable. The immediate survivors succumbed within the first 24 hours after surgery. Late survivors lived from 31 hours to at least 2 years. The majority of the operative deaths in the first two groups resulted from bleeding either from the anastomosis or the chest. Three of the 5 dogs (Groups 1 and 2) which survived operation succumbed to ventricular fibrillation secondary to infarcted ventricular muscle. Operative deaths

TABLE II. RESULTS OF SURGERY

GROUP	NO.	OPERATION	OXYGENATOR	OPERATIVE DEATHS	IMMEDIATE SURVIVORS	LATE SURVIVORS	NORMAL SINUS RHYTHM RE-ESTABLISHED
1 and 2	15	Coronary cut and reanastomosed	Melrose and Baxter	10	5	2	11
3 and 4	16	Direct anastomoses	Baxter and Björk	7	9	4	14
5	19	Anastomoses via graft	Björk	12	7	4	19

TABLE III. CAUSES OF DEATH

OPERATION	NUMBER	OXYGENATOR	BLEEDING ANASTOMOSES	BLEEDING ELSEWHERE	PERFUSION	CENTRAL NERVOUS SYSTEM	THROMBOSIS AT THE SITE OF ANASTOMOSIS	INEFFECTUAL BEAT	MISCELLANEOUS
Division and reanastomoses	15	Melrose and Baxter	3	5	1	0	0	0	2 fibrillation 2 infarct 1 mediastinal emphysema
Left subclavian to left coronary direct	16	Baxter and Björk	1	2	1	4	0	3	1 torn vena cava 1 lost 1 empyema 3 unknown cause
Left subclavian to left coronary via graft	19	Björk	3	1	1	0	3	9	1 tamponade 1 pneumonia after angiography

in Groups 3 and 4 resulted from central nervous system damage due to inadequate brain perfusion, from stenosis at the anastomosis and from bleeding. Immediate survivors died from central nervous system damage or bleeding. There were 4 late survivors: 2 died with patent anastomoses, one of unknown cause and one of empyema; one was lost and one is living with an electrocardiogram suggesting myocardial infarct during the first postoperative week.

In Group 5, 9 of the dogs died on the table because the ventricular beat was too weak to support life. Whether this was the result of myocardial damage from long perfusion or an inadequate anastomosis we could not determine. No animal perfused more than 140 minutes or with arrest for more than 100 minutes survived the operation (Fig. 1). Bleeding from the anastomosis accounted for 3 operative deaths. Three dogs died during the first 24 hours: one of tamponade, 2 from shock with patent anastomoses. Of the 4 late survivors, 3 developed thrombosis at the suture line and died. One succumbed to pneumonia on the seventh postoperative day following angiograms. The anastomosis was patent.

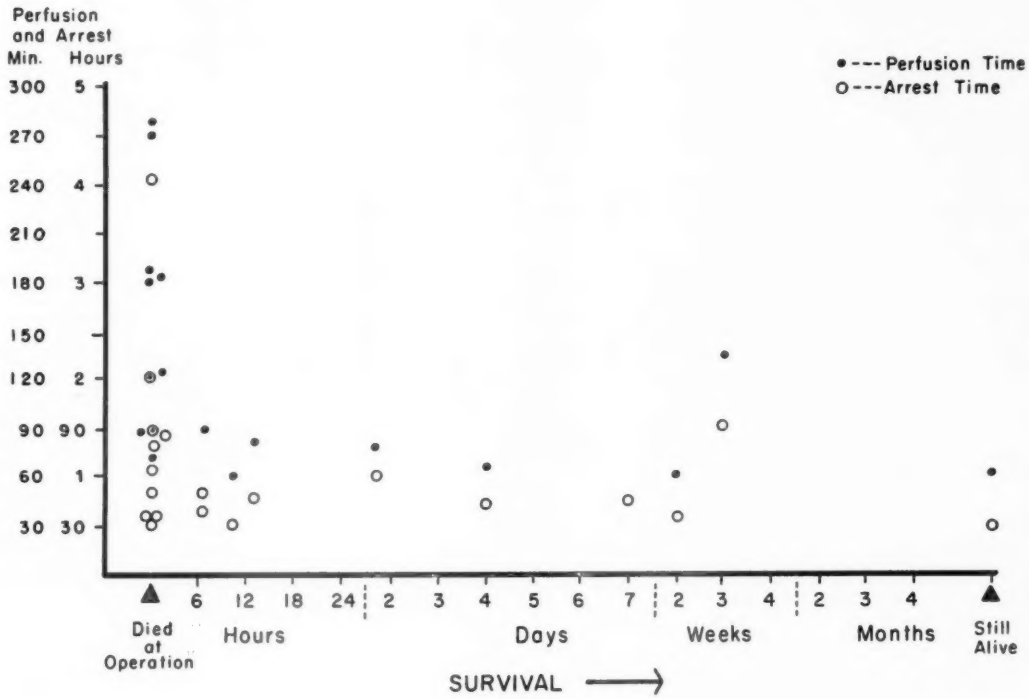

Fig. 1.

DISCUSSION

Since we were chiefly interested in the left coronary system a left thoracotomy was employed routinely. Any postoperative collection of blood was evacuated and the chest tubes were not removed until the chest was dry.

The anastomosis was made as simply as possible (Fig. 2). In the smaller vessels, an end-to-end anastomosis was made over a stent (plastic or copper

wire) with 6-0 interrupted silk sutures. For larger dogs in which the coronary branches were of better size, a continuous over-and-over suture or vertical mattress suture appeared satisfactory. In some of the animals, a sleeve-type anastomosis was made. This sleeve worked quite well in attaching a lyophilized graft to the subclavian stump, but ordinarily the coronary vessel was itself too small to receive such a cuff and a simple interrupted suture anastomosis was employed in attaching the graft to the coronary.

In all but 6 of the 50 dogs subjected to potassium arrest a sinus rhythm was restored. In Groups 1 and 2 where a simple division and reanastomosis of the coronary artery were performed, release of the aortic tape usually resulted in spontaneous return of the heart to normal sinus rhythm.

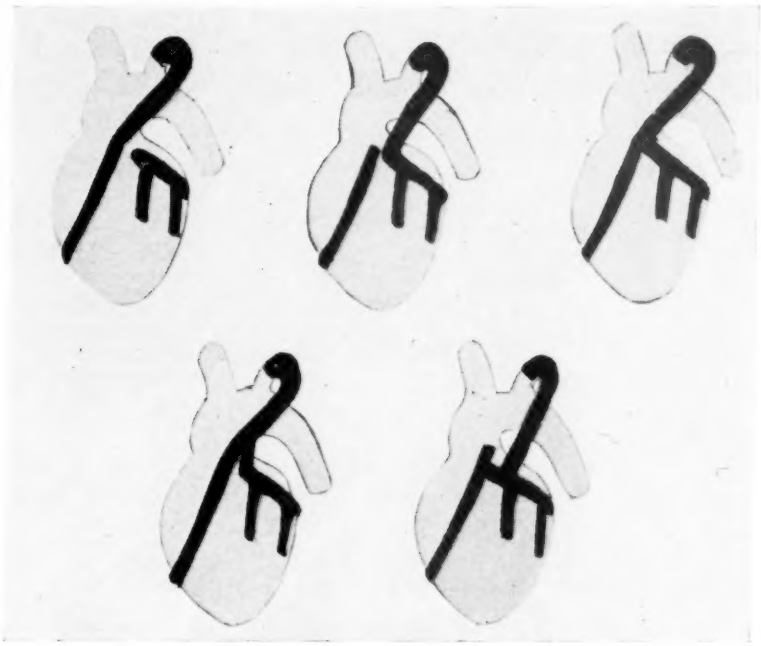

Fig. 2.—Types of anastomoses (left to right).
1. Left subclavian to anterior decending.
2. Left subclavian to circumflex.
3. Left subclavian to left coronary.
4. Left subclavian to left coronary via graft.
5. Left subclavian to circumflex end to side.

In Groups 3, 4, and 5, where the subclavian artery was anastomosed to the left coronary artery, a normal sinus rhythm was seldom achieved spontaneously on release of the occluding tapes.[7] This was the result of at least two factors: first, the time of arrest was long; and, second, as one part of the left ventricle received blood from the grafted artery and the other from the remaining coronary, there was a difference in perfusion and thus in recovery rate of the corresponding muscle. On several occasions we have seen one ventricle fibrillate while the other maintained a sinus rhythm. Ventricular fibrillations were converted to normal sinus rhythm with electroshock. This conversion was simple

and required but one shock except in rare instances. In 2 dogs, instead of fibrillation, a state of tetany of the entire left ventricle resulted after the occluding tapes were removed.

Bleeding from the anastomosis itself resulted in the death of several dogs, although small leaks would disappear once the heparin had been canceled with protamine sulfate. Postoperative bleeding, except from the anastomosis, was only an occasional problem and was felt to be purely a technical error. We kept a log of blood loss but tended to estimate the postoperative blood volumes of the animals on the basis of performance and size of the heart, after sinus rhythm had been restored and the animal was ready to be removed from the machine. The average postoperative blood loss ran between 300 and 400 c.c. and was usually adequately replaced with one pint of citrated fresh dog blood.

Within the limits of our experimental use, all three oxygenators were satisfactory when operated by an experienced team.

SUMMARY

Fifty dogs were subjected to surgery of the coronary arteries while being perfused with a pump-oxygenator and with cardiac arrest induced with potassium citrate. The coronary arteries were divided and resutured or were anastomosed to the left subclavian artery and its branches, either directly or through an interposed freeze-dried graft. In nearly all experiments a normal sinus rhythm could be restored, although ventricular fibrillation often occurred first. Not all hearts could be restored after prolonged periods of arrest, and in some the beat was too weak to support life. Such deaths may have been related to myocardial damage associated with the citrate arrest.[13]

The cardiac catheter introduced into the subclavian artery was first used to inject potassium citrate and to obtain simultaneous coronary angiograms; then as a stent in suturing the artery, and finally to inject contrast medium to check on the patency of the anastomosis.

Late survivors were few. The problems of suture anastomosis of small vessels were not adequately solved in these experiments.

We would like to express our appreciation to Dr. H. Brodie Stephens, San Francisco, for his criticisms and suggestions.

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Pediatric Surgery

CONDUCTED BY MARK M. RAVITCH, M.D.

THE ANATOMY OF THE PELVIC AUTONOMIC NERVOUS SYSTEM IN CASES OF HIGH IMPERFORATE ANUS

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AMONG the many anatomic structures that subserve rectal continence, two of the most important are the pelvic autonomic nerves and the anal sphincter muscles. The purpose of this paper is to describe the pattern of pelvic autonomic nerve distribution that was found by dissecting the hemipelvis of 3 infants suffering from the high variety of imperforate anus, to compare this with the findings in 7 normal pelves and to discuss the surgical implications of these facts.

High imperforate anus refers to those cases in which the bowel ends blindly or in a fistulous communication with the bladder, prostatic urethra or upper end of vagina at the level of the pelvic floor, or pubococcygeal line of Stephens.[7, 9] It corresponds with the type III of Ladd and Gross.[3]

Various operations are designed to produce in the perineum an opening for the bowel as a substitute for the anus and it is hoped that patients may gain some measure of control over this opening so that it is not merely a perineal colostomy. This catastrophe may be preventable in some cases by greater attention to anatomic detail, and, similarly, the normal function of other pelvic organs, in particular the bladder, will not be endangered.

In order to emphasize the significance of the anatomy of the high imperforate anus, a brief account of the anatomy and physiology of normal rectal control is necessary.

THE NORMAL CONTROL OF DEFECATION

The proper function of the anal sphincters depends upon their integrity and on a nerve reflex arc originating in the anal and rectal mucosa, passing through the sacral region of the spinal cord and ending in the smooth and striated muscles of the internal and external anal sphincters. Cerebral cortical influence considerably modifies the reflexes and, under normal conditions, the call to defecation can be suppressed whenever necessary. Additional

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voluntary muscles, such as the levator ani, may be used to support the action of the sphincters when an emergency arises. These complex processes may be analyzed basically by considering their component parts.

Rectal and Anal Sensation.—Clinical and experimental evidence suggests that removing major portions of the rectum or anesthetizing its mucosa eliminates rectal sensation and interferes with fecal continence. Rectal sensation, however, is undoubtedly a compound of at least three factors: first, actual rectal distention; second, pressure of the fecal mass or the investigator's balloon on the pelvic floor; and, third, sensation from the upper end of the anal canal. Fecal incontinence following major rectal excisions for carcinoma may be due more to the wide excision necessary in this condition with consequent damage to the pelvic plexus and particularly the nerve supply to the levator ani than to actual loss of the rectum. Rectal excision for Hirschsprung's disease is compatible with fecal continence, probably because the dissection is maintained close to the rectal wall, leaving the pelvic floor and other structures in the neighborhood undamaged and also because about 1.5 cm. of rectum is left in situ.

The Nerve Pathway.—Fibers from the rectum anus, and pelvic floor travel through the pelvic plexus and the inferior hemorrhoidal nerves and then via the pelvic parasympathetic and third and fourth sacral spinal nerves to the sacral cord. Here, they synapse with other components of the reflex arc and with fibers from the brain which effect voluntary control. Efferent impulses return by the same route to the smooth muscle of the rectum and internal anal sphincter and the striated muscle of the external anal sphincter and levator ani.

The Sphincter Muscles.—There are two anal sphincters the internal being a condensation of the circular muscle of the rectum with the external of striated muscle situated around it. Their action is reinforced by that part of the levator ani which forms a sling under the rectum at the anorectal junction. This is the puborectalis muscle which is attached to the undersurface of the pubis and whose fibers intermingle with those of the deep part of the external anal sphincter. By contracting, it pulls the anorectal junction forward increasing the angulation and closing the rectum. Little mention is made of this muscle by those who have investigated the physiology of rectal control. It is difficult to study because of its position, but it is very evident that it plays an important role. It cannot maintain a state of strong contraction for long periods as, like all striated muscle, it is subject to the phenomenon of fatigue; but it can augment the action of the external anal sphincter when the need arises. If for any reason the external sphincter is absent or ineffective, the puborectalis can maintain continence provided that the feces are not fluid.

The muscles which produce rectal continence are, therefore, the internal and external anal sphincters and the puborectalis.

THE CONTROL OF DEFECATION IN PATIENTS TREATED FOR A HIGH IMPERFORATE ANUS

Having briefly noted the mechanism of normal rectal control, it is necessary to apply this mechanism to the patient treated for a high imperforate anus.

Anal and Rectal Sensation.—In the majority of cases the anal canal is absent and there can never be anal sensation. Rare instances occur in which the proctodeum is present and, if it is not damaged at operation, sensation from it may be appreciated and considerably influence the development of rectal control.

The lowest portion of the terminal bowel is usually extraperitoneal, and at operation this peritoneum must be divided to expose the bowel or the fistula if present. According to Wood Jones,[11] that part of the rectum which represents the hind-gut extends from Houston's first valve, which is intraperitoneal to the third valve which is at the level of the peritoneal reflection. Beyond this, the rectum represents postallantoic gut. Goligher and Hughes[2] demonstrated that rectal sensation can be appreciated when a balloon is inflated in the colon 15 cm. from the anus. Therefore, in the high imperforate anus a short length of the terminal bowel should represent the upper segment of the definitive rectum and should be capable of producing some rectal sensation. As this is an important feature of the defecation reflex, the whole terminal bowel should be preserved during the surgical correction of this anomaly.

The Afferent and Efferent Pathways.—These will be discussed in the section on anatomy.

The Sphincter Muscles.—The external anal sphincter develops independently of the rectum and some fibers may be present in the high imperforate anus. In the absence of a proctodeum, however, it is poorly developed and forms only a thin sheet similar to the platysma, lying in the subcutaneous tissue over the perineum. Its action is likely to be insignificant because it is weak and because it becomes involved by the scar tissue which inevitably forms to some extent in the perianal region as a result of surgery.

The internal anal sphincter is developed from the circular muscle fibers of the lower end of the rectum and will therefore be absent.

The levator ani is the only muscle which can act as a sphincter in the high imperforate anus, and Stephens[7] has drawn attention to the variations in its anatomy in different rectal anomalies. Sacral agenesis is frequently associated with imperforate anus,[8, 10] and, if present, other pelvic structures including the levator ani are malformed. With three or less sacral vertebrae this deficiency is severe.

Thus, whatever hope there may be for the development of some degree of rectal continence in the patient who has had a high imperforate anus will depend upon a short length of bowel with an intact nerve supply, and upon the levator ani. The former will provide the sensation of rectal filling and the latter, the sphincter mechanism.

THE ANATOMY OF THE PELVIC AUTONOMIC NERVES IN THE NORMAL INFANT

Material and Method.—The specimens were from 5 male newborn infants. Death was due to various causes, such as prematurity, esophageal atresia, duodenal atresia, and multiple congenital cardiac anomalies. The pelvic organs

in all cases were entirely normal with the exception of a week-old baby with one kidney hypoplastic and the other grossly hydronephrotic. The seminal vesicles in this case were remarkably dilated.

The half pelvis including the whole rectum and bladder was removed at autopsy, fixed in 10 per cent formol-saline, and dissected in stages. At the completion of the dissection, the specimen was photographed and blocks were cut from the anorectal region, the bladder, prostate, pelvic plexus, and autonomic nerve bundles, and histologic sections made. These sections were stained with hematoxylin and van Gieson's stain and examined to confirm the presence of nerve fibers.

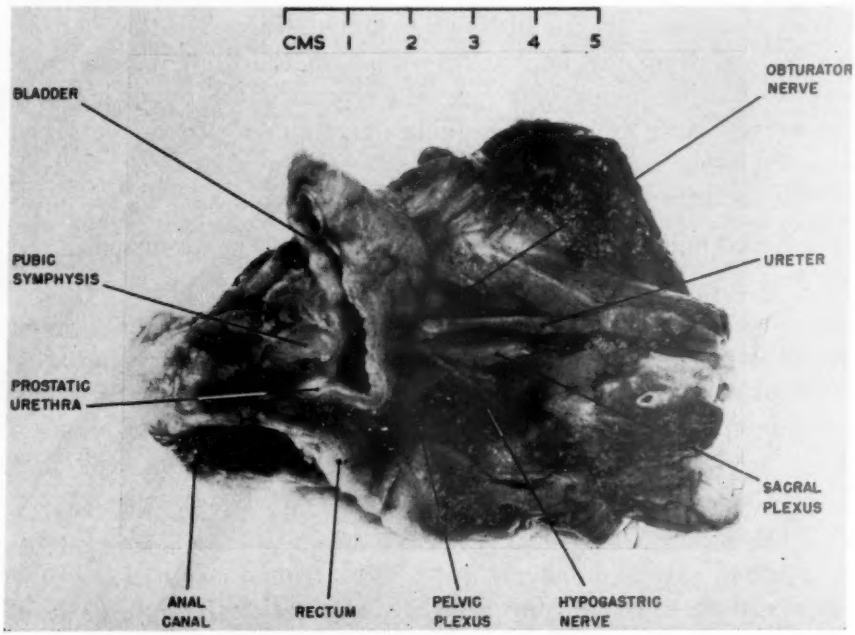

Fig. 1.—Normal right hemipelvis viewed from medial side. The rectum has been retracted to reveal the pelvic plexus.

Findings (Figs. 1-4).—In general, the anatomy of the autonomic nerves was similar to that in Mitchell's[5] description except that no contribution to the pelvic plexus from the second sacral nerve was found in any of the specimens. This feature was also described by Caria Mendes.[1] The hypogastric nerve (Fig. 5), carrying sympathetic fibers, originates in the prelumbar plexus below the common iliac artery and travels toward the side wall of the pelvis accompanying the internal iliac artery. It lies immediately beneath the peritoneum and is vulnerable, particularly on the left side during dissection of the superior hemorrhoidal vessels at the brim of the pelvis. As it travels downward and forward, it sends branches to the sigmoid colon and rectum which turn medially and upward crossing the mesenteric vessels. Just before it enters the pelvic plexus at the ureterovesical junction, it gives off another branch which turns laterally to accompany the vas. The lower end of the ureter receives branches from the hypogastric nerve and from the pelvic plexus.

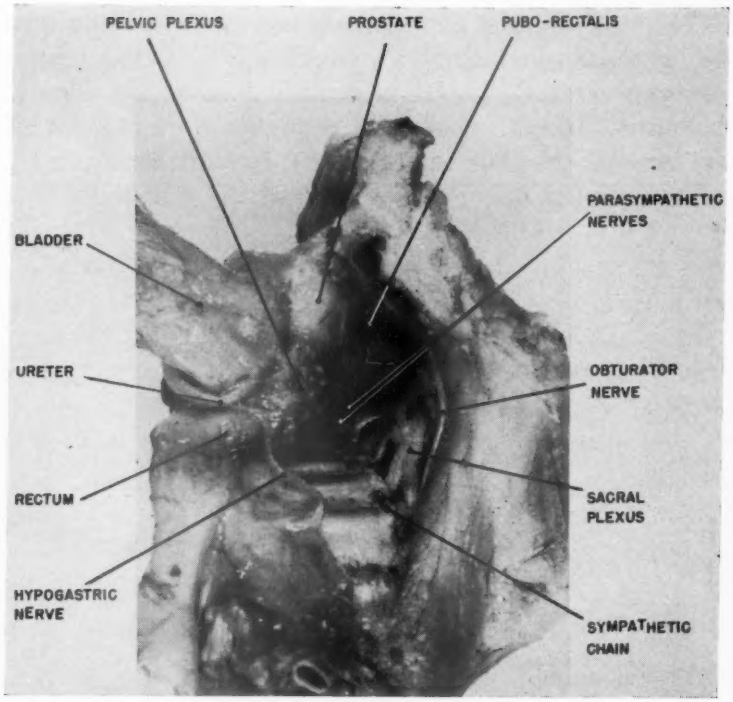

Fig. 2.—Normal right hemipelvis viewed from above. Shows parasympathetic nerves joining pelvic plexus.

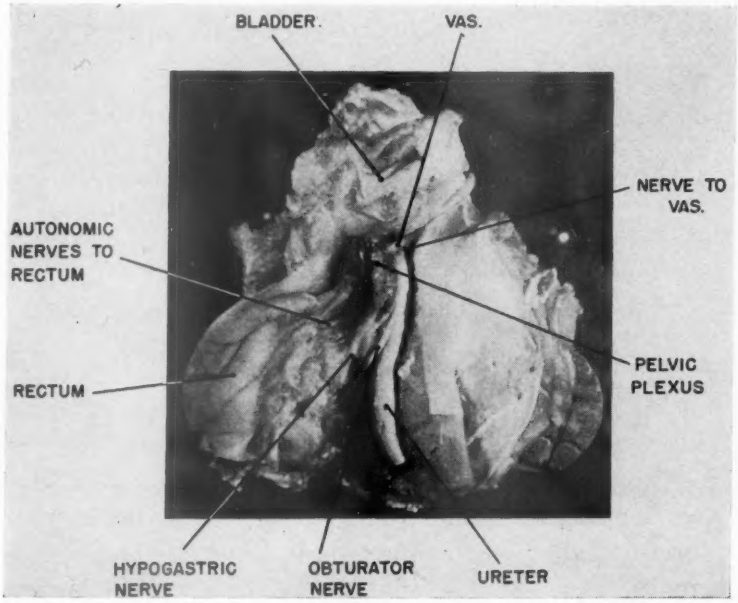

Fig. 3.—Normal right hemipelvis viewed from above. Branches of hypogastric nerve to sigmoid and rectum.

The pelvic plexus (Fig. 6) consists of a meshwork of nerve fibers and ganglion cells covering the side wall of the pelvis and extending from the inferolateral surface of the bladder and prostate anteriorly to the posterolateral surface of the rectum posteriorly. It is separated from the dense fascia which covers the obturator internus, pyriformis, and levator ani muscles by lymphatics, fat, and blood vessels. Medially, it is covered by peritoneum and pelvic fascia. There are many ganglion cells in the areas where nerve fibers from the plexus enter the bladder and prostate, particularly in the cleft between the two organs posteriorly and around the termination of the vas and vesicles (Fig. 7).

The rectum is innervated with sympathetic fibers from the hypogastric nerves and parasympathetic fibers from the pelvic plexus. The two sets of fibers become intermingled and enter the posterolateral surface of the rectum as far inferiorly as the anorectal junction. (Fig. 8.) Removing the rectum with a surrounding layer of pelvic cellular tissue would inevitably damage the posterior part of the plexus.

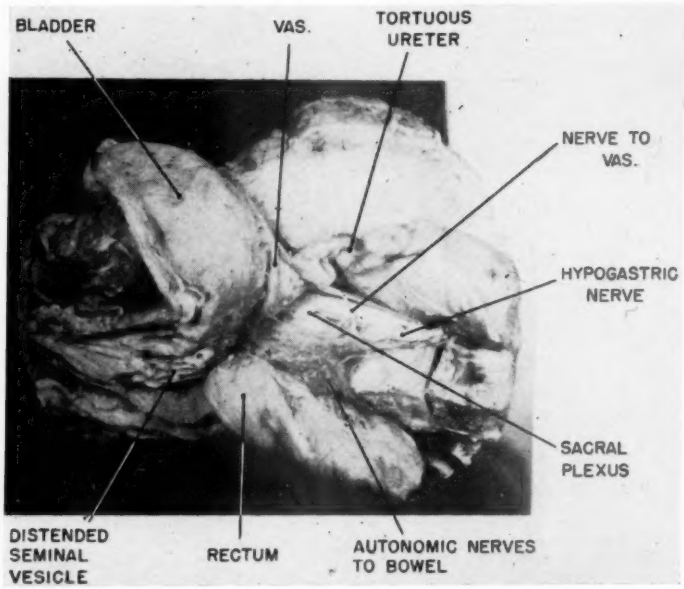

Fig. 4.—Right hemipelvis with normal rectum but grossly dilated seminal vesicles.

The sacral sympathetic chain passes downward and forward lying on the anterolateral surface of the sacral vertebral bodies. Fibers from the third and fourth ganglia enter the pelvic plexus with the parasympathetic nerves.

The parasympathetic nerves (Fig. 9) emerge as two leashes from the third and fourth anterior sacral foramina in company with the spinal nerves and immediately turn medially to enter the lateral aspect of the pelvic plexus which is covered on its inner side at this point by the rectum. The bigger leash arises from the third sacral nerve. No separate contribution from the second sacral nerve was seen in any of the specimens despite careful dissection. Disturbances of micturition which occasionally follow an excessively wide rectal excision in the depths of the pelvis may be caused by damaging these nerves.

ANATOMY OF THE PELVIC AUTONOMIC NERVES IN THE HIGH IMPERFORATE ANUS

The specimens dissected were all male and will be described individually.

CASE 1.—This infant was found at birth to have a high imperforate anus and a transverse colostomy was performed. It died of multiple cardiac anomalies when one week old.

Fig. 5.

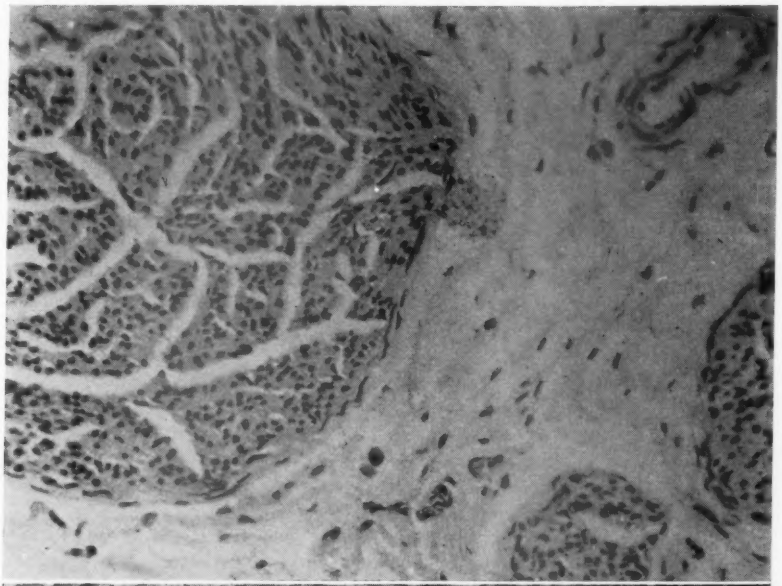

Fig. 6.

Fig. 5.—Histologic section through hypogastric nerve. Shows a loose collection of nerve bundles.

Fig. 6.—Histologic section through pelvic plexus. Shows meshwork of nerve fibers and ganglion cells.

At autopsy the deformity proved to be one of the common variety of high imperforate anus with a rectourethral fistula (Fig. 10). The terminal bowel which, despite a transverse colostomy, was still grossly distended at the time of death, narrowed

abruptly as it approached the base of the bladder forming a fistulous tract which passed forward and upward through the prostate to enter the urethra. There were five sacral vertebrae.

Fig. 7.

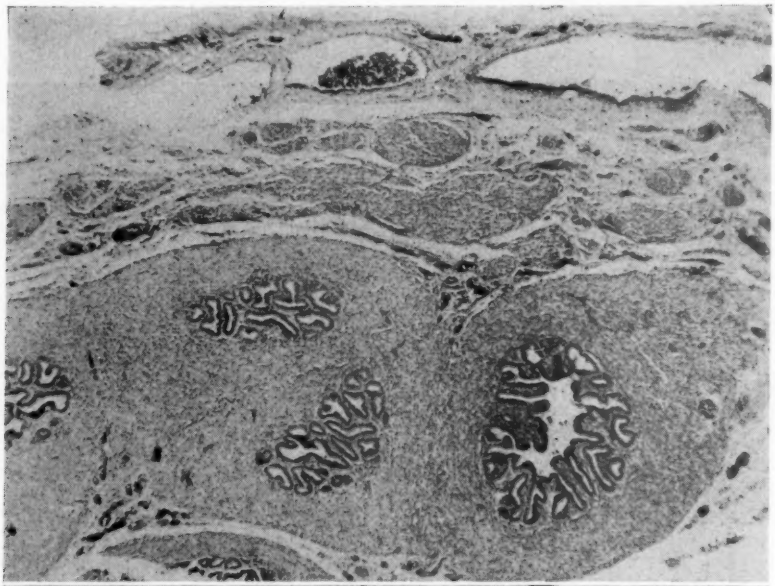

Fig. 8.

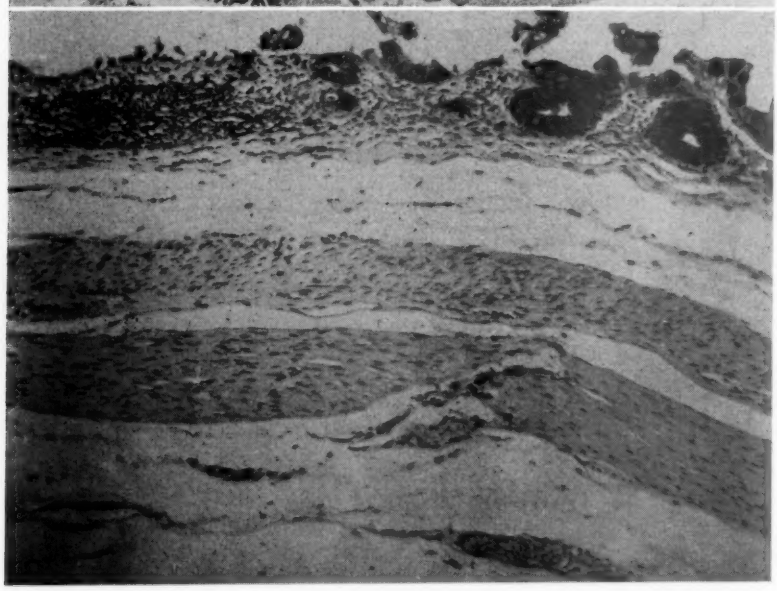

Fig. 7.—Histologic section showing nerve fibers and ganglion cells in neighborhood of seminal vesicle.

Fig. 8.—Histologic section through wall of normal rectum. Shows nerve fibers in the areolar coat.

The hypogastric nerves followed the same course as in the normal pelvis, sending branches medially to the bowel and entering the pelvic plexus at the ureterovesical junction. The lower end of the ureter received fibers from the hypogastric nerve and the pelvic plexus.

The pelvic plexus extended from the ureterovesical junction to the lower border of the prostate and sent many fibers into the lateral and posterior aspects of the bladder and prostate along this line of attachment. Some fibers, intermingled with those from the hypogastric nerve, turned upward from it to supply the lower end of the bowel. In

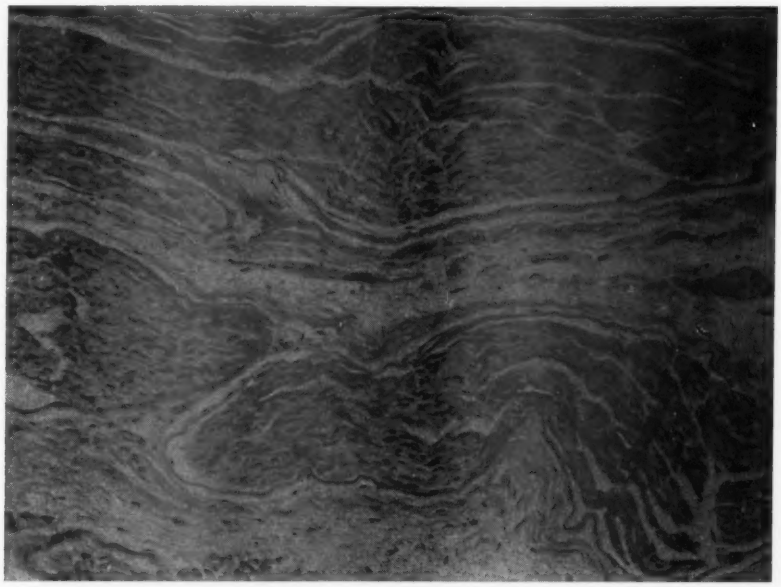

Fig. 9.—Histologic section through pelvic parasympathetic nerves.

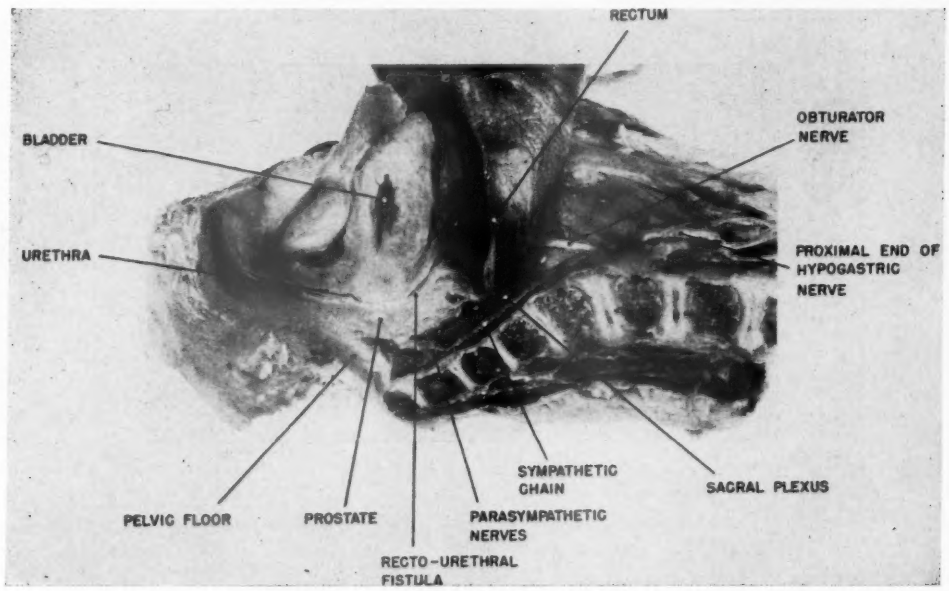

Fig. 10.—Medial view of hemipelvis of Case 1. Imperforate anus with rectourethral fistula.

the absence of the rectum the posterior extent of the plexus was considerably smaller than normal and merged with the soft tissues on the posterior surface of the fistula tract and prostate. Laterally, the pelvic plexus received two leashes of parasympathetic fibers

from the third and fourth sacral spinal nerves and a contribution from the sacral sympathetic chain. Medially, it was closely related to the extraperitoneal part of the terminal bowel.

As in the normal pelvis, the parasympathetic nerves appeared as two leashes, one large and one small, from the third and fourth anterior sacral foramina (Fig. 11) and entered the lateral side of the pelvic plexus a short distance below the point at which the

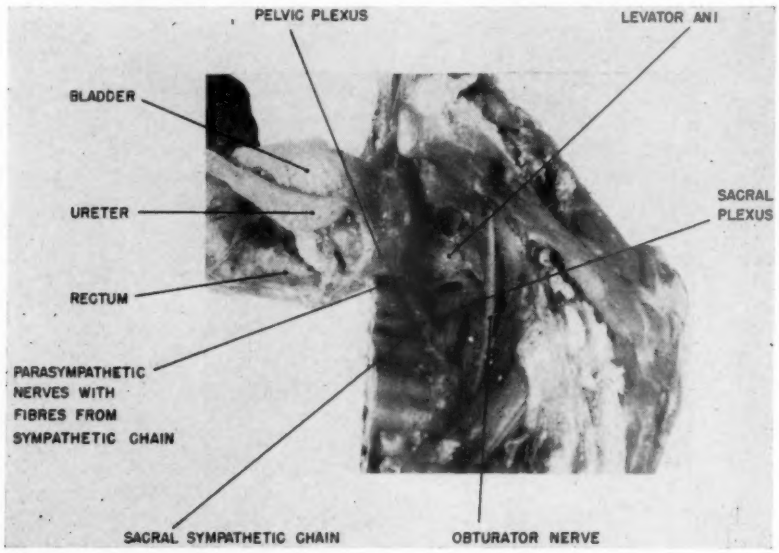

Fig. 11.—Hemipelvis of Case 1 viewed from above. Shows parasympathetic nerves entering pelvic plexus.

Fig. 12.—Histologic section through bowel wall in neighborhood of fistula in Case 1, showing nerve fibers in the areolar tissue.

bowel narrowed to form the fistula. The nerves of the two sides were closer together anteriorly than normal as there was no rectum between them.

The sympathetic chain followed the normal course sending fibers from its lower ganglia to accompany the parasympathetic nerves to the pelvic plexus.

The terminal 1 cm. of bowel was extraperitoneal and possessed a rich nerve supply derived from the hypogastric nerves and pelvic plexus. The fistula tract was buried in soft tissue at the base of the bladder and the prostate, and on histologic section many nerve fibers and ganglion cells were seen surrounding it and the posterior and lateral surfaces of the prostate (Fig. 12).

The levator ani curved beneath the lower end of the prostate distal to the fistula and being attached posteriorly to the tip of the fifth sacral vertebra formed a complete diaphragm for the posterior part of the pelvis. The puborectalis was represented by the anterior sling of muscle fibers beneath the prostate and urethra.

The anatomy of the pelvic autonomic nerves in this case therefore differed from the normal only as much as might be expected from the absence of the rectum. The terminal bowel and fistula tract were found to be richly supplied with both sympathetic and parasympathetic fibers.

CASE 2.—The infant was born with a high imperforate anus and died at the age of one day following a transverse colostomy. Autopsy revealed that the bowel ended blindly in the pelvis and, though it was attached to the base of the bladder, no fistula communicating with the bladder or urethra was found (Fig. 13). The sacrum had five vertebrae. The pelvic peritoneum was damaged at the time the pelvis was removed from the body, and it was not possible to define the whole course of the hypogastric nerve. The distal end, however, was still visible as it entered the pelvic plexus at the ureterovesical junction.

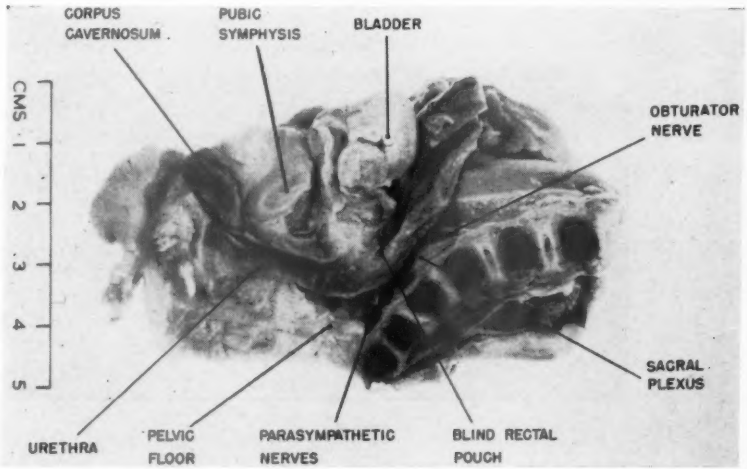

Fig. 13.—Medial view of hemipelvis of Case 2. Imperforate anus without fistula.

The pelvic plexus extended from the ureterovesical junction to the prostate but was more closely applied than in the normal. Many nerve fibers could be seen turning upward from it to the terminal bowel while others ran directly into the bladder and prostate. Because of the absence of the rectum, the extent of the plexus was limited posteriorly. Laterally, it received the parasympathetic nerves and some fibers from the sacral sympathetic chain and, medially, it was related to the terminal bowel for a short distance.

The parasympathetic nerves appeared in company with the sacral spinal nerves and entered the undersurface of the pelvic plexus below the blind end of the bowel (Fig. 14). As in the previous case, the nerves of the two sides were closer together than normal.

The sympathetic chain did not differ from the normal and provided some fibers which entered the pelvic plexus.

Histologic examination revealed many nerve fibers running through the areolar tissue covering the wall of the terminal bowel. Other fibers and ganglion cells were seen in the connective tissue and muscle between the bladder and bowel and were particularly numerous around the ureteric orifices, seminal vesicles, and prostate.

The levator ani covered the space between the urethra and the sacrum as in Case 1 although the central portion was not as well developed. The puborectalis was represented by that part which looped beneath the urethra as a sling. This case also proved that the terminal bowel was richly supplied with nerve fibers.

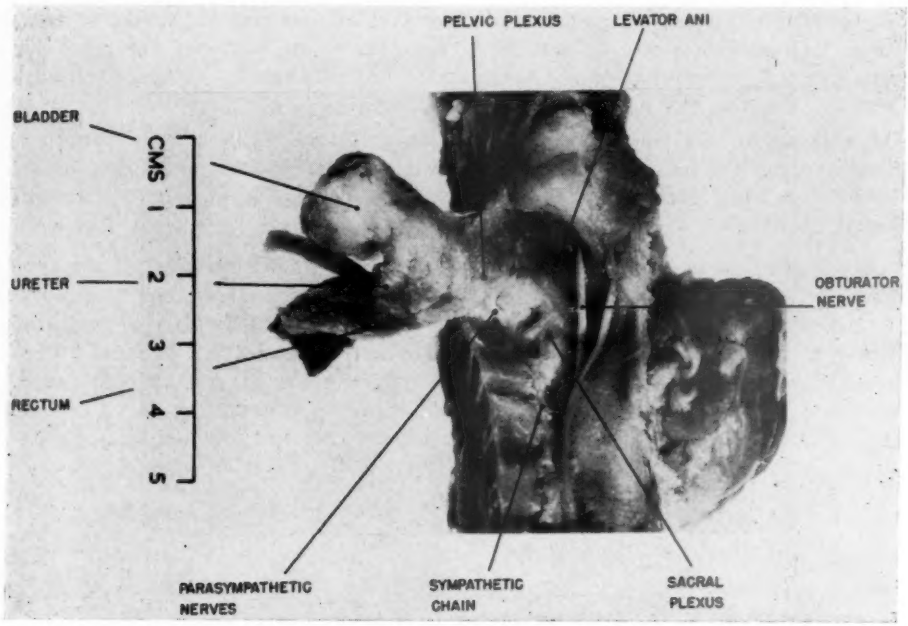

Fig. 14.—Hemipelvis of Case 2 viewed from above showing parasympathetic nerves entering pelvic plexus.

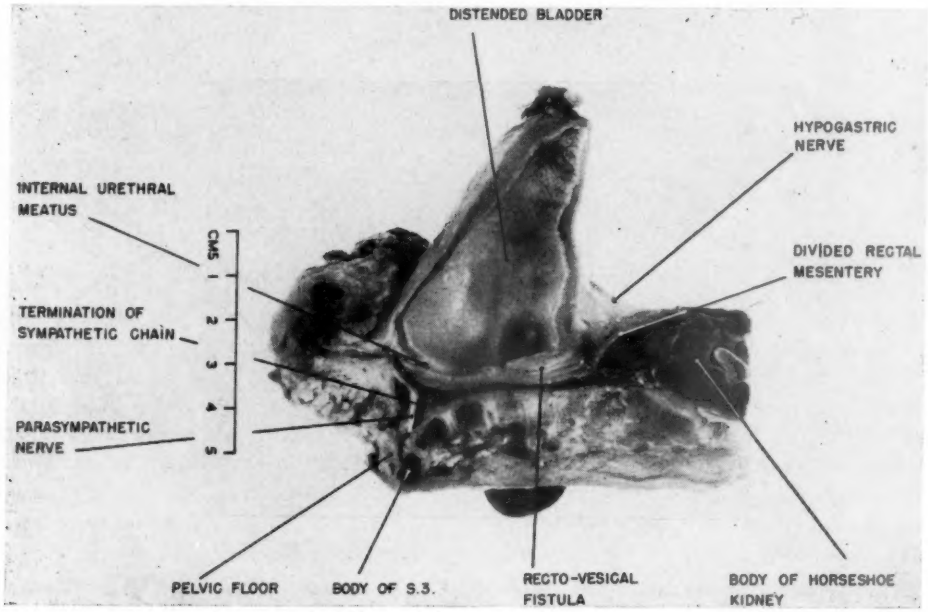

Fig. 15.—Medial view of hemipelvis of Case 3. Imperforate anus with rectovesical fistula, three sacral vertebrae, and distended bladder.

CASE 3.—This infant had a high imperforate anus with a rectovesical fistula. At operation on the first day of life, a transverse colostomy with ligation and division of the

fistula was performed but the infant succumbed shortly afterward. At autopsy, apart from the divided fistula, the pelvic organs were intact. The bladder was grossly distended with a hypertrophic wall and there were only three sacral vertebrae. There was a horseshoe kidney lying across the lower lumbar region (Fig. 15).

The terminal bowel was removed and examined separately after its mesentery was divided (Fig. 16). Histologic examination of the mesentery (Fig. 17) revealed nerve fibers passing through it, and these were also seen in the subserosal layer of the bowel wall, being particularly numerous in the region of the fistula.

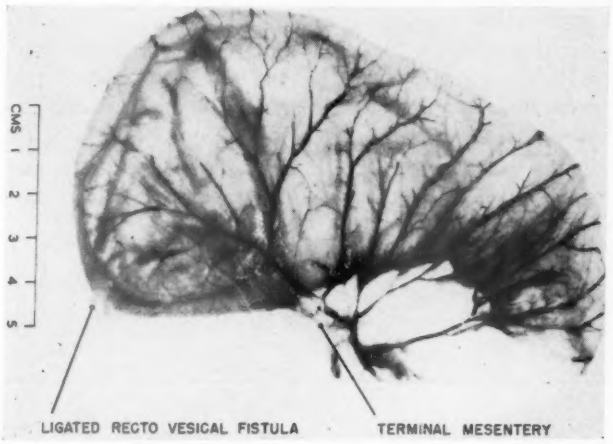

Fig. 16.—Terminal bowel from Case 3.

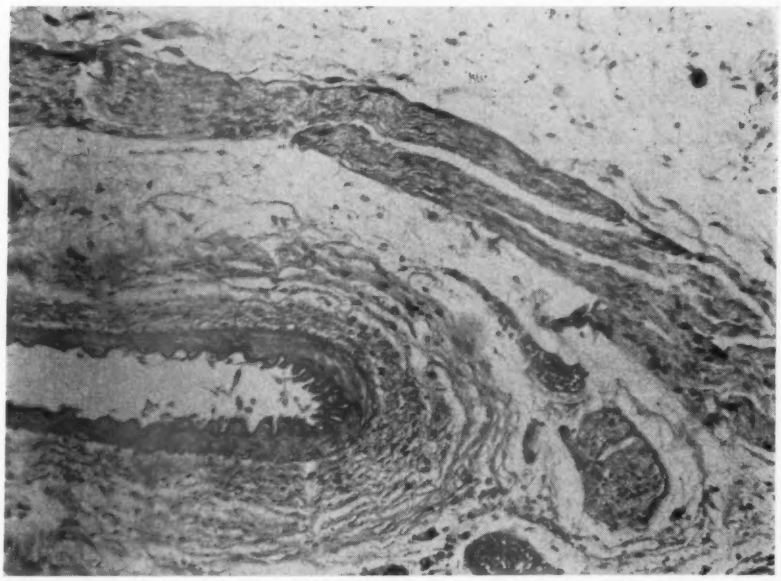

Fig. 17.—Histologic section through mesentery of terminal bowel in Case 3. Shows nerve fibers in the neighborhood of a blood vessel.

The hypogastric nerve crossed the body of the horseshoe kidney, traveling laterally to the side wall of the pelvis while giving branches medially to the bowel. It entered the pelvic plexus at the ureterovesical junction.

The pelvic plexus was considerably distorted by the grossly distended bladder and was applied so closely to its lateral surface that no plane of separation could be found between the two. It extended from the ureterovesical junction to the prostate and received one

leash of parasympathetic fibers at its lower end. There was no mechanical obstruction such as urethral valves which accounted for the megacystis, so presumably it was due to a functional abnormality associated with the sacral agenesis.

Only one leash of parasympathetic fibers arising distal to the third sacral vertebra was found, and this joined the pelvic plexus on the inferolateral surface of the bladder. The deficiency in parasympathetic nerves was accounted for by the sacral agenesis (Fig. 18) which also caused the nerves of the two sides to lie very close together at their origin, there being no fourth vertebral body to separate them.

The sympathetic chain passed down on the front of the sacral vertebral bodies and could not be traced any further than the third.

Histologic examination of the prostate and the wall of the bladder revealed many nerve fibers and ganglion cells particularly in the area surrounding the rectovesical fistula. The nerve elements were situated in the adventitial coat of the bladder and some fibers could be seen penetrating the muscle layers.

The muscles of the pelvic floor were poorly formed and, although some elements of the levator ani were present, it is doubtful whether they could have functioned as an efficient rectal sphincter.

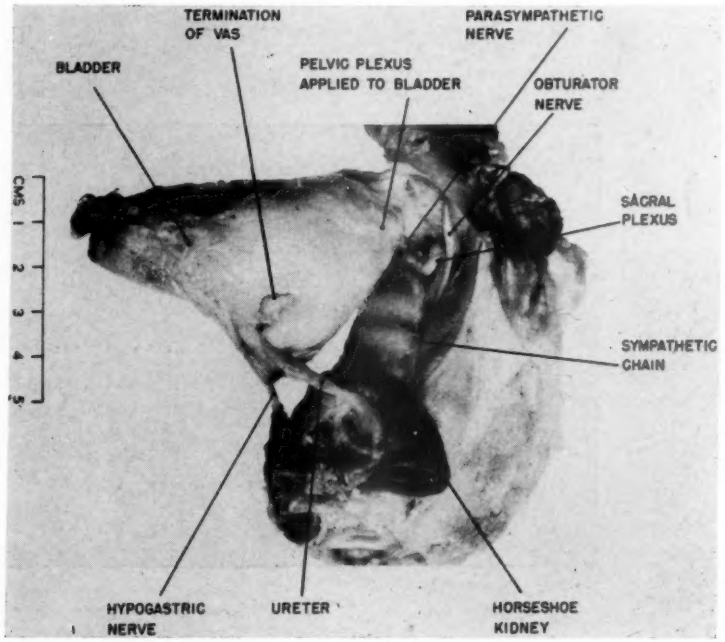

Fig. 18.—Hemipelvis of Case 3, viewed from above, showing single pelvic parasympathetic nerve and pelvic plexus applied to the bladder wall.

DISCUSSION

Nerve elements with a general distribution similar to the normal were found in the three cases of high imperforate anus and the abnormalities that were present could be accounted for by the rectal anomaly. In addition, the third case revealed some of the anatomic and functional effects of severe sacral agenesis.

In all cases the terminal bowel was supplied, via the pelvic plexus, by the pelvic parasympathetic and hypogastric nerves. The main nerve supply for the bladder, seminal vesicles, and prostate entered along their posterolateral surfaces

as in the normal. The presence of nerve fibers and ganglion cells in an organ does not necessarily indicate that the organ will function normally as was exemplified by the third case in which the bladder was grossly distended but possessed a plentiful supply of ganglia. This consideration may also affect the function of the terminal bowel. However, if this bowel is to possess some of the motor and sensory functions associated with the normal rectum, every effort should be made to preserve its nerve supply.

SURGICAL IMPLICATIONS

Having noted some of the anatomic features of the high imperforate anus, it is necessary to apply them to the operative treatment of the condition.

If the most distal end of the terminal bowel is derived from the hind-gut and is capable of producing rectal sensation, then no part of it should be removed. Mobilization of the terminal bowel should be kept to an absolute minimum and be carried out with great care so as to preserve its nerve supply as far as possible. Just sufficient mesentery and peritoneal attachments should be divided to allow the bowel to be brought down to the perineum without mobilizing more than necessary. Some nerves must inevitably be damaged, but it is possible that limited regeneration may occur.[4, 6]

The tunnel through the pelvis for the bowel should be formed with caution. It should commence as far posteriorly as possible to avoid the nerve supply to the base of the bladder and seminal vesicles, and should be created by blunt dissection exactly in the midline to avoid damaging the pelvic parasympathetic nerves which are close on either side. It should if possible pass in front of the puborectalis sling so that this muscle may act as a sphincter. Stephens[8] has suggested a posterior approach to facilitate this maneuver; but, if special care is taken during the combined abdominoperineal approach, the muscle may be identified and separated from the urethra and the lower border of the prostate.

Other factors must be considered if a severe degree of sacral agenesis is discovered during the preoperative clinical and radiologic examination. Bladder function will probably be abnormal and the pelvic floor poorly formed. The pelvic parasympathetic nerves will be particularly vulnerable when the tunnel for the bowel through the pelvis is being made and damage to these structures will further jeopardize bladder function. The prognosis for both urinary and fecal continence in such a case would be extremely poor.

Advancing the terminal bowel to the perineum with minimal mobilization is easier shortly after birth when the bowel is in its most distended and elongated state. If, after an initial colostomy, the definitive operation is performed when the child is older, the terminal bowel is collapsed and its mesentery is short, increasing the difficulty of obtaining sufficient length to bring down to the perineum. Mobilizing the bowel dividing fistulas, and identifying the pelvic floor are best carried out by a combined abdominoperineal approach, and this operation should be performed as an immediate procedure provided that the infant's general condition is satisfactory.

MANAGEMENT

The surgical treatment of the imperforate anus is difficult and fraught with many disappointments. Only those surgeons who have special training and experience in operating on these small patients and who are prepared to supervise the long period of bowel training which is usually required should undertake their management. Parents must be encouraged to persevere with the unpleasant tasks that are often required of them, and the surgeon must place himself at their disposal at all times to give advice and hope. Only the special pediatric surgical centers can provide the expert and efficient nursing care which is so valuable in these cases, and with modern communications babies can be transported rapidly and safely. There is no greater tragedy than the child condemned to a lifetime of misery and embarrassment by errors in the initial surgical treatment and lack of interest in the after care.

SUMMARY

The anatomy of the autonomic nerves in the normal pelvis is described and compared with three cases of high imperforate anus. The terminal bowel in each case is found to have a rich autonomic nerve supply.

When compared with the normal, the development of rectal control in patients treated for a high type of imperforate anus may depend on the terminal bowel, its nerve supply, and the levator ani muscle. Greater care and attention to anatomic detail will preserve these structures and also the normal function of other pelvic organs.

I would like to thank Mr. H. H. Nixon of the Hospital for Sick Children, Great Ormond Street, London, and Dr. Orvar Swenson of the Boston Floating Hospital, Boston, for their help and encouragement. Also, Dr. Martin Bodian of the Hospital for Sick Children and Dr. H. Edward MacMahon of the New England Medical Center, Boston, for the autopsy material which was used for dissection. Thanks are also due to the laboratory and photographic technicians of both hospitals for the microscopic sections and photography; and finally, The Commonwealth Fund in New York for the Fellowship which enabled me to continue this work in the United States.

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Editorials

CONDUCTED BY I. S. RAVDIN, M.D.

Diverticulosis and Diverticulitis

Colonic diverticulosis and diverticulitis are fraught with far greater potential hazards than many realize. Their complications have been well recognized for many years. Yet the tradition of avoiding operation until it is forced upon us has fostered an attitude which seems to prevent rational thinking about diverticular disorders and their pathologic potential, to a point where we are like the ostrich with his head in the sand—perhaps surrounded by dangers, but feeling quite secure in the failure to perceive them.

The frequency of complications and the tendency of the inflammatory process itself to recur at frequent intervals are well known. Yet until recently even surgeons have avoided operation until this was forced upon them. Surgeons who readily resect other diseased organs are still prone to regard colonic diverticulitis as basically a "medical disease," with operation reserved for complications if or when they occur. For some unaccountable reason, diverticulitis seems to bear a "hands off" sign for a surprisingly large number of our colleagues.

There are some indications that the medical profession is becoming more aware of the need for elective operative interference in many cases of diverticular disease of the colon. Several excellent papers have emphasized this point. Standard textbooks of surgery still reflect the "conservative" attitude that "medical" treatment is indicated for colonic diverticulitis, but most of the recent editions do refer to a trend toward more frequent operative interference aimed at prevention of complications. It is also gratifying to note emphasis upon the value of a diverting colostomy as an emergency measure in the presence of acute diverticulitis (for rest is fully as important in the treatment of inflammation in this area as in any other). One recent British text reflects a real awareness of the lethal potential of this disease by these words: "Literally, a diverticulum means a wayside house of ill-fame, and these wayside houses certainly live up to their evil reputation."

Hemorrhage, one of the most serious of all the complications of diverticular disease of the colon, has been so little recognized that a considerable segment

of the profession even denies its occurrence. A review of the literature reveals that moderate bleeding occurs in between 10 and 30 per cent of the reported cases of diverticulitis. Massive exsanguinating hemorrhage, on the other hand, has not been recognized generally as a major complication of diverticular disease. There are many cases recorded in which this has occurred, and at least one author gave a graphic description of blood spurting from the orifice of a diverticulum observed through a sigmoidoscope. Numerous cases have been reported in which recurrent hemorrhage has been stopped by resection of the colon, the resected segments showing only diverticula. Histologic evidence of bleeding from erosion of diverticular vessels has been obtained. Injection studies have revealed the presence of good-sized arteries in the neck of diverticula, confirming previous anatomic studies based upon other techniques. Rives pointed out that severe hemorrhage is more likely to occur in diverticulosis, in contrast to the more moderate bleeding associated with diverticulitis. Despite all of the evidence to the contrary, one still finds many who say that if hemorrhage occurs it must be coming from carcinoma, polyps, or other colonic disease. These conditions do indeed cause rectal bleeding, but it is rarely of such severity as to threaten acutely the patient's life. In our experience diverticulosis is far and away the most common cause for exsanguinating rectal hemorrhage.

Colonic resection is now a relatively safe procedure with minimal morbidity and low mortality. There is reason to believe that the mortality from untreated or inadequately treated diverticulitis is considerably higher than that following resection. Recurring diverticulitis is a disabling disease, each attack fraught with the hazard of many lethal complications. Further, patients can bleed to death as rapidly and as surely from an eroded vessel in the neck of a colonic diverticulum as they can from a vessel of comparable size anywere else in the body. In view of all these considerations, the pleas of Welch, Waugh, and many others for a more aggressive attitude toward recurrent diverticular disorders of the colon is amply justified. It is high time that internists and surgeons alike recognize the fact that one should be as ready to remove this kind of disease as any other, and that the results should be as good.

In conclusion, one could do no better than to quote the editorial comment of the late Evarts Graham, reviewing a series of abstracts on this subject in the 1953-1954 *Yearbook of Surgery:* "The general tone of all the preceding five articles is for early resection of the involved colon in a case of diverticulitis. This attitude is thoroughly sound in view of the comparative safety of the operation nowadays. The old policy of watchful waiting is becoming untenable."

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Surgical Residency Training and Independent Responsibility

Surgical proficiency has been obtained in a variety of ways. Many have acquired their surgical ability more or less on their own. Others have been initiated into the problems of surgery by a relatively brief hospital service. Still others have served a preceptorship. The first serious effort to prepare thoroughly experienced surgeons were the German and Austrian systems of clinic assistantship. Undoubtedly, the greatest advance was made when Professor Halsted initiated the residency training program which has proved such a vital force in improving the caliber of surgical practice, research, and teaching. The type of program he developed has been widely applied, sometimes following the precise concepts and principles he considered important, sometimes with wide modification.

With this diverse background it is natural that opinions concerning the training of surgeons should vary. Factors which govern these opinions include the individual's own past training, opportunities available to him for training others, and his own philosophy toward surgery in general. Not long ago Dr. George Crile, Jr.,[2] commented editorially on certain aspects of this problem. With some of his statements I am in wholehearted agreement; with others I am in sharp disagreement. Since the problem is so important and since I am certain that Dr. Crile would welcome expression of another point of view by a colleague and friend, I shall record my own attitude.

Dr. Crile pointed out the confusion which exists between training and experience. He was certainly right when he stated, "If a resident, without supervision, attempts to perform an operation before he has assisted on enough operations to know the technic, his time is wasted. If he is assisted in doing an operation before the basic pattern of the indications and technic has been mastered, time is wasted by both resident and assisting staff surgeon." No one could logically argue for unsupervised and uncontrolled responsibility and operative opportunities beyond the competence of the trainee. I judge that Dr. Crile ascribes relatively little importance to independent responsibility and opportunity for independent operative experience in the training program. With this I do not agree. Dr. Crile states: "When this training is complete, the surgeon can devote his life to acquiring experience." It would be a pity, to be sure, if he did not continue to improve his judgment, his concepts, and his technical ability throughout his surgical practice. It seems far preferable, however, that he begin practice with as adequate experience as he can possibly obtain. Dr. Crile feels that much of the confusion between training and experience has arisen from the phrase "independent responsibility." To me this phrase connotes a truly important aspect of the training program and refers, not to unsupervised operative opportunities at too early a stage, but rather to independent responsibility when the individual has progressed to the point where he is able to accept and profit from it.

During the years of surgical residency training, the trainee anticipates advice, encouragement, and stimulation from the supervising staff. He learns

much, not only from the chief of service and his associates, but from those on the resident staff who are more experienced than he. Gradually, but more quickly than one might anticipate, he acquires judgment, initiative, and operative dexterity. Though advice should be constantly available to him, his growth and maturation should not be strangled by too much of it. In my opinion, these young men can be thoroughly trusted. They can handle many problems entirely on their own and ultimately practically all of them. Rare is it that they take advantage of a generous attitude toward their gradual assumption of such responsibility. During the final year of training the resident surgeon demonstrates remarkable capacities. I know of nothing more gratifying than to see year after year these young men assume this final mantle of responsibility and demonstrate thorough proficiency in the clinics, on the wards, and in the operating rooms. It is a joy indeed to find them dexterous surgeons, sound in judgment, capable of performing the most difficult surgical procedures.

The sort of training program which Professor Halsted began at The Johns Hopkins is interpreted in different ways by different individuals. Concerning this matter, Dr. Crile states: "The Halsted system of residency training has been held up as an ideal to which residency training programs should aspire. There is no question that this system gave to the one surgeon who emerged as the chief resident an unparalleled educational opportunity. It is often forgotten, however, that the Halsted resident spent seven to ten years in the hospital and that, 'independent responsibility' came only after training was complete. The chief resident in the Halsted system was the equivalent of a junior staff surgeon. After full training he stayed to do research, teach and gain clinical experience. Following this, he was fitted to become a professor. Unsuccessful aspirants in the Halsted type of pyramidal residency were given three or four years of good training, but they had little experience and no independent responsibility before they were dropped. Yet these men emerged as the well-trained surgeons who have built the reputation of the Halsted system." In 1952 Carter[1] wrote about the Halsted residency program under the title, "The Fruition of Halsted's Concept of Surgical Training." He pointed out that there has become widespread the impression that Halsted's resident surgeons served for many years both as assistant resident and as resident surgeon but that actually of the 17 resident surgeons trained by Halsted, 2 served 7 years each, 4 approximately 5½ years, 5 approximately 4½ years, and the other 6 lesser periods of time. The 17 residents who completed their training in the 33 years during which Halsted directed the program, departed from the institution well-trained surgeons. They left at a time when there was a crying need for them as teachers and as heads of services. Seven of them became professors of surgery; one, surgeon-in-chief of a hospital; one, professor of clinical surgery; three, associate professors of surgery; one, instructor in anatomy; and four, private practitioners. Fifty-five men served under Halsted as only assistant resident surgeons. They certainly did well also. Fifteen of them became professors; 5, clinical professors; 5, associate professors; 3, assistant professors; 5,

assistant clinical professors; 8, instructors; and 14, private practitioners. A sizable number went into the surgical specialties: orthopedic surgery, roentgenology, urology, otolaryngology, neurosurgery, and one into medicine. Quite a few of those who remained in the field of general surgery completed their residency experience elsewhere or at The Johns Hopkins after the unfortunate death of Professor Halsted. They certainly made their weight felt in the progress of American surgery. Approximately 75 per cent of both groups—those who were Halsted's resident surgeons and the larger group who were only his assistant resident surgeons—obtained university posts. Both they and those who limited their work to the private practice of surgery were excellent practitioners and lived up to the ideals which Dr. Halsted held high.

Only a relatively small number of surgeons can aspire to university teaching positions. The vast majority will find no such opportunities. Everyone who directs a surgical training program hopes that some of his residents will become teachers and investigators as well as practitioners of surgery. He realizes, however, that many will confine their work to the private practice of surgery. He has the same hopes for all of them—that they will not only be good surgeons but that they may make some significant original contributions and that they will, if they can, pass on to others the opportunities which they themselves have had.

It seems evident to me that the fruition of a good training program should be a final period as resident surgeon with ample opportunities for individual responsibility in and out of the operating room. I should think that the young surgical aspirant would try to obtain a position in a training program where he would have the benefit of advice and encouragement from a staff whom he respects, where he would have every opportunity for individual self-development and from which he might hope to emerge as a thoroughly well-trained, experienced surgeon. I would suppose he would enter training not so much to emulate his teachers as to develop his own capacities under their guidance. I believe that it must be such thoughts that have prompted the Conference Committee on Graduate Training in Surgery to advise that programs be set up in such a manner as to afford such operative opportunities to the resident. I also believe that the same considerations have prompted the American Board of Surgery to suggest that candidates for Board certification obtain training in a program which includes a final year of senior responsibility.

If Professor Halsted were alive I feel confident he would be gratified at the widespread extension of the type of residency training he initiated. I do not imagine he would be at all concerned that such programs vary from one institution to another, that the length of training differs as does the scope of surgery covered, and that some maintain a pyramidal system while others have abolished it. I do believe he would be gratified that so many are designed to give the resident surgeon the independent responsibilities and opportunities he considered important.

I have expressed my views without regard to the opportunities available and I have not considered the problems which are arising as the result of

increasing hospital and professional service insurance coverage. We cannot, however, face these problems wisely unless we define for ourselves the objectives of the residency training program.

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Surgical History

CONDUCTED BY JOHN R. PAINE, M.D.

EPONYMIC FRACTURES: ROBERT WILLIAM SMITH AND SMITH'S FRACTURE

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ADAMS, Cheyne, Graves, Stokes, and Colles were the stellar attractions of the medical schools of nineteenth century Dublin, but no constellation is complete without its lesser stars. The star of Robert William Smith, anatomist, surgeon, pathologist, has not shone with the brilliance of his more famous colleagues, but after a scrutiny of his accomplishment, it becomes apparent that the fault is ours, not his. Counting as his friends some of the best medical minds of the time, he was accepted as an equal; among distinguished teachers, he was unsurpassed. His name is perpetuated as an eponym for an unusual fracture of the lower end of the radius, while his description of neurofibromatosis is almost forgotten.

FRACTURE OF THE LOWER EXTREMITY OF THE RADIUS, WITH DISPLACEMENT OF THE LOWER FRAGMENT FORWARD[6]

This is an injury of exceedingly rare occurrence, and one which presents characters closely resembling those of dislocation of the carpus forward. It generally occurs in consequence of a fall upon the back of the hand, and the situation of the fracture is from half and inch to an inch above the articulation; it is accompanied by great deformity, the principal features of which are a dorsal and a palmar tumour, and a striking projection of the head of the ulna at the posterior and inner part of the fore-arm; the dorsal tumour occupies the entire breadth of the fore-arm, but is most conspicuous internally, where it is constituted by the lower extremity of the ulna displaced backwards; from this point, the inferior outline of the tumour passes obliquely upwards and outwards, corresponding in the latter direction to the lower end of the superior fragment of the radius. Immediately below the dorsal swelling there is a well-marked sulcus, deepest internally below the head of the ulna, directed nearly transversely, but ascending a little as it approaches the radial border of the fore-arm.

The palmar is less remarkable than the dorsal tumour; formed principally by the lower fragment of the radius, it is obscured by the thick mass of flexor tendons which cross the front of the carpus, but towards the ulnar border of the limb there is a considerable projection, which marks the situation of the pisiform bone, passing down to its attachment into which can be seen the tendon of the

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flexor carpi ulnaris thrown forwards in strong relief. The transverse diameter of the fore-arm is not much altered, but the antero-posterior is considerably increased, and the radial border of the limb becomes concave at its lower part.

In the case from which the preceding drawing was made, the patient, in endeavoring to save himself from being run over by a car, fell with great violence upon the back of the hand; the lower end of the radius was broken and driven forwards along with the carpus, and the head of the ulna was displaced backwards.

I cannot speak with accuracy as to the anatomical characters of the injury, having never had an opportunity of examining after death the skeleton of the fore-arm, in those who had during life met with this accident; nor is there any preparation showing the exact relative position of the fragments, in any of the pathological collections in Dublin; but still I feel satisfied that the injury, the external characters of which have been just described, is a fracture of the lower end of the radius, with displacement of the lower fragment along with the carpus forwards, and of the head of the ulna backwards, and that it has not unfrequently been mistaken for dislocation of the carpus forwards and of the bones of the fore-arm backwards.

Fig. 1.—A lateral radiograph of a wrist showing Smith's fracture, a fracture of the lower end of the radius with displacement of the distal fragment forward.

The description is clear. There is no question that the fracture is located in the same region as that of Colles, but the direction of the displacement is reversed (Fig. 1). There should be no confusion between these fractures, and the fracture dislocations of the wrist described by John Rhea Barton.[4] It comes as something of a surprise, therefore, to find that Watson-Jones[12] considered Smith's fracture nearly always to be a fracture dislocation, that is, Barton's fracture, and to see a classical example of Barton's fracture illustrated under the title of Smith's fracture. *Indignor quandoque bonus dormitat Homerus** (Horace). Thomas[11] has fallen into this same error, for, in classifying Smith's fractures into three types, Type 2, a fracture dislocation, is certainly not a Smith's fracture at all.

The *Treatise on Fractures in the Vicinity of Joints and on Certain Forms of Accidental and Congenital Dislocations*[6] (1847) in which the description of Smith's fracture appeared was the most important work on fractures by a British author to follow the great monograph of Astley Cooper on *Dislocations*

*Even the worthy Homer sometimes nods.

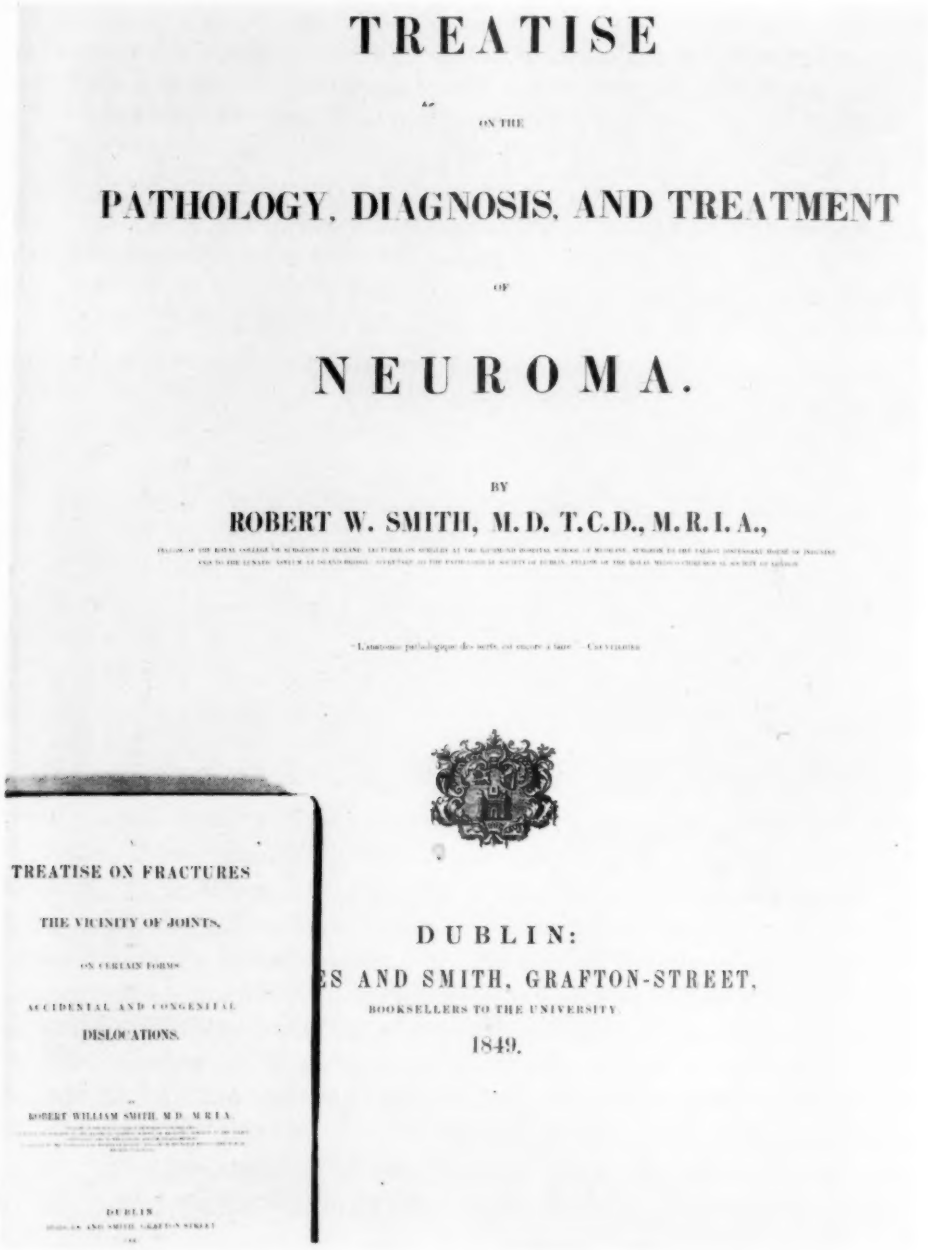
and Fractures in the Vicinity of Joints (1822). The initial chapter, "On the Diagnosis and Pathology of Fractures of the Neck of the Femur," is outstanding. Smith disagrees with Cooper as to the relative amounts of shortening which accompanies intra- and extracapsular fractures and shows that initially the amount of shortening with extracapsular fractures is greater. In patients with old ununited intracapsular fractures, however, he found large amounts of shortening, and attributes Cooper's error to the fact that he based his opinion on the measurement of such cases. In a period in which our knowledge of these fractures is based primarily on their radiographic characteristics, the detailed reports of their pathologic appearances as demonstrated by extensive dissection can still be perused with profit. The introduction of statistical data into medical literature is noted here in Smith's tables listing his own experience with 28 extracapsular, 28 intracapsular, and 4 impacted fractures of the hip, and in an analysis of 140 specimens of hip fractures in various Dublin museums.

In the chapter "On Fractures of the Bones of the Forearm in the Vicinity of the Wrist Joint," Smith corrects Colles' original description by placing the site of Colles' fracture more distally. "The situation of the fracture is not so high as Mr. Colles states it to be; I have never seen it more than an inch above the carpal end of the bone; in the majority of cases it is not so much." There are good anatomic plates illustrating this injury as well as separations of the distal radial epiphysis.

In 1849, only 2 years after the appearance of his *Treatise on Fractures in the Vicinity of Joints,* Smith's other great work was published. *A Treatise on the Pathology, Diagnosis, and Treatment of Neuroma*[8] was indeed a most unusual book. It was said to be the largest book printed in Ireland up to that time; an elephant folio measuring 48 by 70 cm. (Fig. 2). The plates were "drawn from life and on stone" by the Irish illustrator Connolly, and are superb. In addition to an extensive review of the literature, Smith presented, among others, the history, course, and pathologic findings in 2 patients with neurofibromatosis. In the first case, a 35-year-old man (Fig. 3), over 800 individual neuromas were demonstrated by dissection. The second case, a 32-year-old man, was found to have 1,400 neuromas, with an estimated additional 600 not dissected. The illustrations of the gross pathology of these 2 cases surpass anything contained in the classical report of von Recklinghausen made 33 years later.[5] Exactly why Smith chose to present his work in such an unwieldy form is difficult to understand since the book fits neither the pocket, the briefcase, nor the bookshelf.

Robert William Smith (Fig. 4) was born in Dublin on Oct. 12, 1807. His father was English; his mother, Irish. Like Colles', Smith's father died early, leaving the family to be brought up by their mother. Both Robert and his brother Sidney, who entered the ministry, received an excellent education. Smith was apprenticed to Dr. Carmichael and studied at several of the Dublin medical schools, principally the Richmond Hospital School. The schools of medicine in Dublin were entering their golden age, and his medical training was as adequate as could be obtained anywhere at that time.

Early in his career Smith taught medical jurisprudence and surgery at the Richmond Hospital School, and was the surgeon for the Lunatic Asylum, Island Bridge. His interest in pathologic anatomy was intense. He was an indefati-



TREATISE

ON THE

PATHOLOGY, DIAGNOSIS, AND TREATMENT

OF

NEUROMA.

BY

ROBERT W. SMITH, M.D. T.C.D., M.R.I.A.,

DUBLIN:

S AND SMITH, GRAFTON-STREET,

BOOKSELLERS TO THE UNIVERSITY.

1849.

TREATISE ON FRACTURES

THE VICINITY OF JOINTS,

ON CERTAIN FORMS

ACCIDENTAL AND CONGENITAL

DISLOCATIONS.

ROBERT WILLIAM SMITH, M.D. M.R.I.A.

DUBLIN

Fig. 2.—The title pages of Smith's *Treatise on Fractures in the Vicinity of Joints* and *Treatise on the Pathology, Diagnosis, and Treatment of Neuroma.* The former is an octavo; the latter a curious elephant folio.

gable collector. He founded the splendid museum of the Richmond Hospital and enriched it from year to year with the fruits of his labors in the form of drawings, casts, and preparations illustrating every phase of surgical pathology.

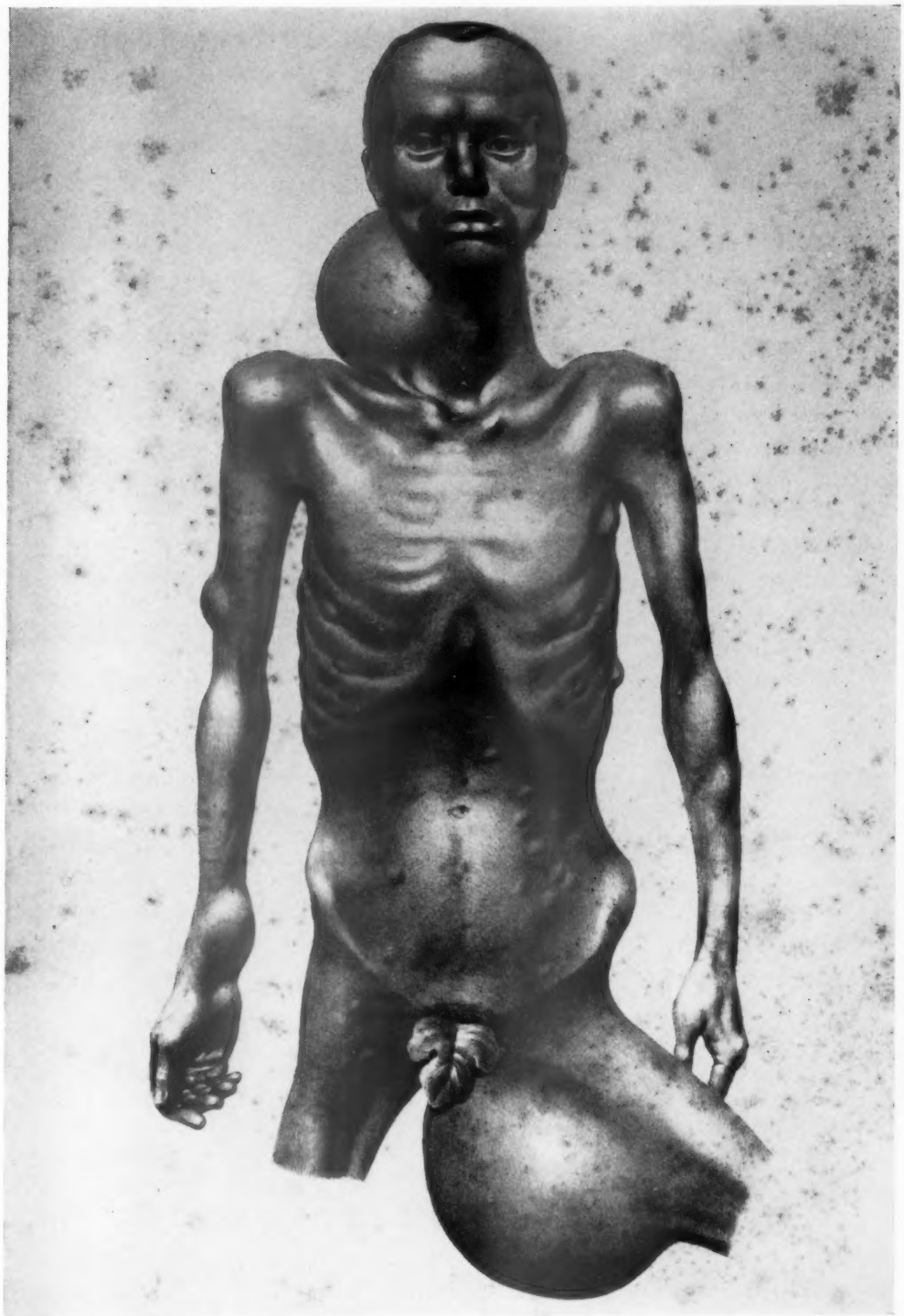


Fig. 3.—Plate I from the treatise on neuroma showing the scale and quality of the illustrations.

In his zeal as a collector, he was compared by his contemporaries with John Hunter himself. After his death, his private osteologic collection was purchased by Trinity College for their Anatomical Museum.

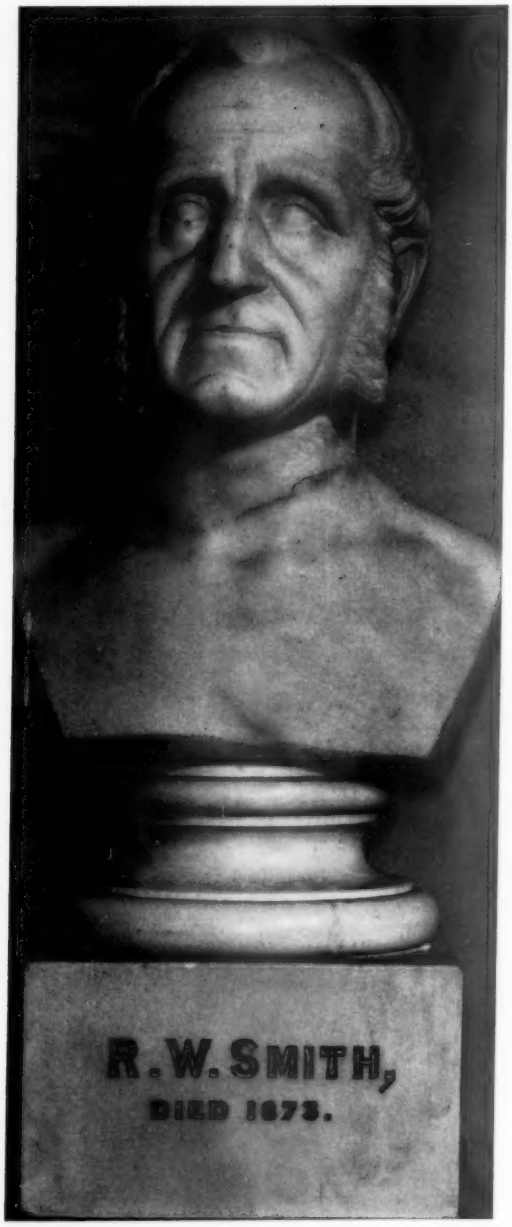


Fig. 4.—Bust of Robert William Smith presented to the Royal College of Surgeons in Ireland by the Pathological Society of Dublin after his death. I am indebted to J. D. H. Widdess, Librarian of the College for the photograph.

In 1838 he was one of the founders of the Pathological Society of Dublin of which he was secretary for 35 years. Associated with him in this enterprise were Colles, Graves, Corrigan, and Stokes. With Stokes, Smith had a more

personal relationship, the two having married sisters. The Pathological Society absorbed his interest and profited from his leadership throughout his life. Almost all of his published works originated as communications to the Society.

His reputation as a pathologist was such that Colles requested Stokes, his physician, to have Smith perform his autopsy, saying that he felt sure that the observations would be made with accuracy and reported with truth. The report of the autopsy was duly made to the Pathological Society of Dublin and later published as a part of Stokes' *Observations on the Case of the Late Abraham Colles, M.D., Formerly Professor of Surgery of the Royal College of Surgeons in Ireland.*[10]

In 1849 Smith became Professor of Surgery at Trinity College, Dublin, a position which he held until his death. He was an enthusiastic teacher and extremely popular with his students. In his later years, he almost completely abandoned practice in order to devote more time to his classes. He was an accomplished linguist, and his reading among ancient medical authors and contemporary French, German, and Italian writers was encyclopedic. In his treatise on fractures, he points out a passage in Dupuytren's *Lecons Orales* in which Dupuytren had been misquoted by his transcribers, an error which was corrected in later editions. Smith's references in his published works to foreign writers exceed in scope those of most British authors of today. Because of his cosmopolitan interests, Smith became an ordinary, corresponding, or honorary member of many medical societies at home and abroad.

In 1873 Smith became Vice-President of the Royal College of Surgeons in Ireland, and undoubtedly would have succeeded to the Presidency had not death intervened after a short illness on Oct. 28, 1873.

It is difficult to clothe with flesh the bare bones of bibliographic data available. His writings reveal Smith to be thorough, observing, and appreciative of fine distinctions. His style is clear and simple, free of affectation. His wit and candor lie hidden except for a rare comment such as the footnote to the bibliography of the treatise on fractures:

> To those who are familiar with the literature of impacted fractures of the neck of the femur, it may, perhaps, appear strange that no reference has been made in the text to the memoir of M. Robert upon the subject, a report upon which was published in the *Bulletin de l'Académie Royale de Médicine,* 1844-45. It was unnecessary to allude to it, inasmuch as, in all that relates to the symptoms, diagnosis and anatomical characters of the injury, the memoir of Robert is merely a translation of that which I published upon the subject in 1840, in the *Dublin Medical Journal.*

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References: 1. Fremont, R. E.; Luger, N. M.; Surks, S. N., and Kleinman, A.: **A.M.A. Arch. Surg.** 68:44, Jan., 1954. 2. Agress, C. M., and Binder, M. J.: **Am. Heart J.** 54:458, Sept., 1957.

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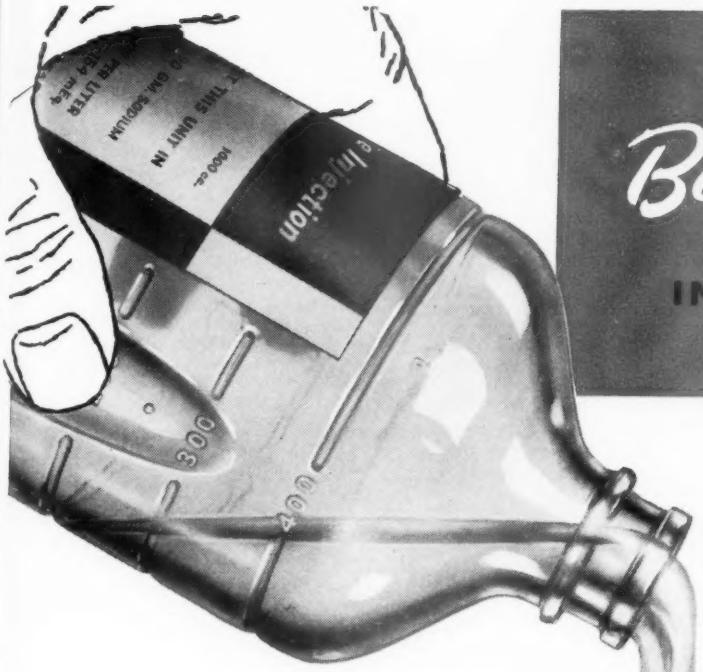

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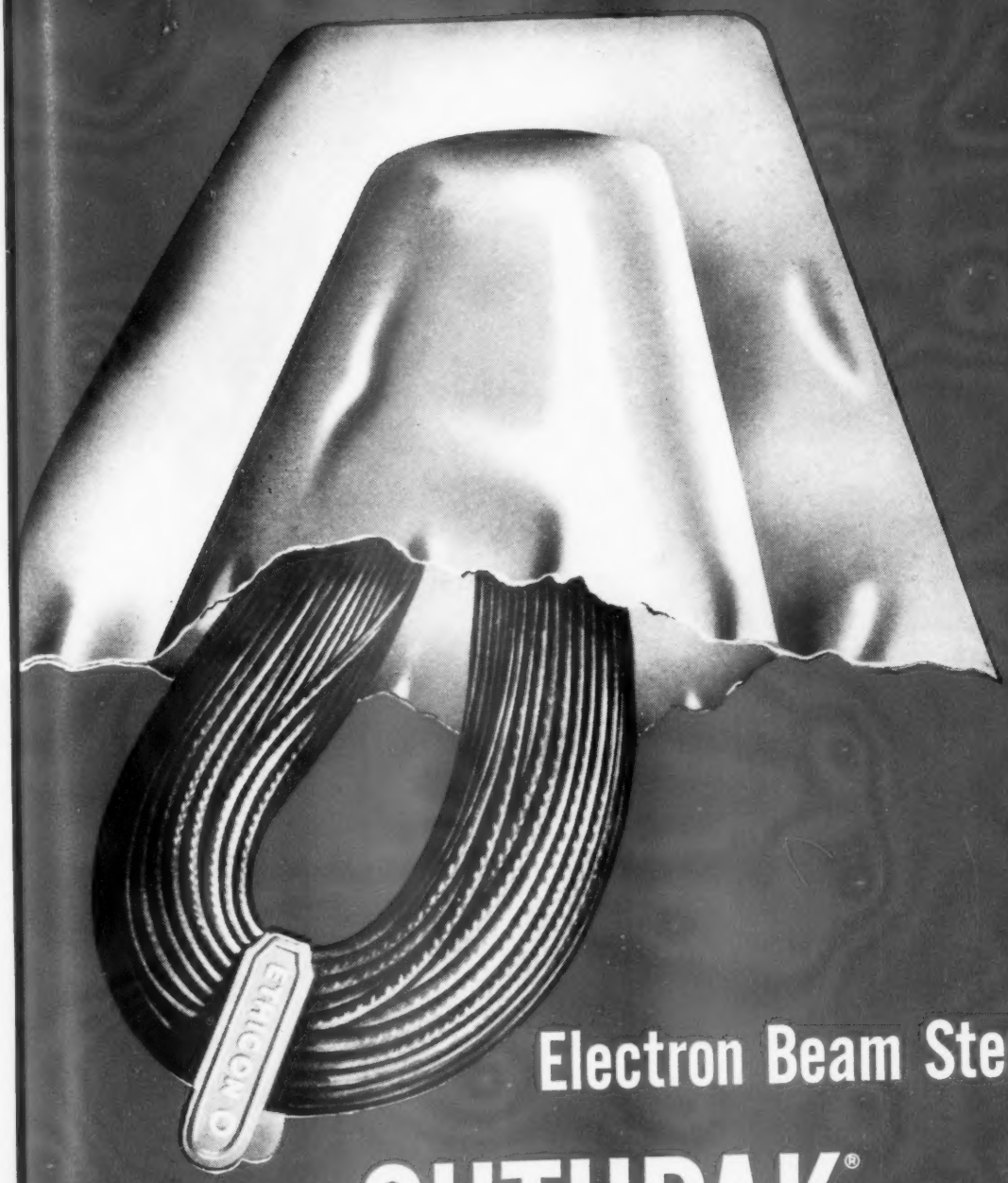

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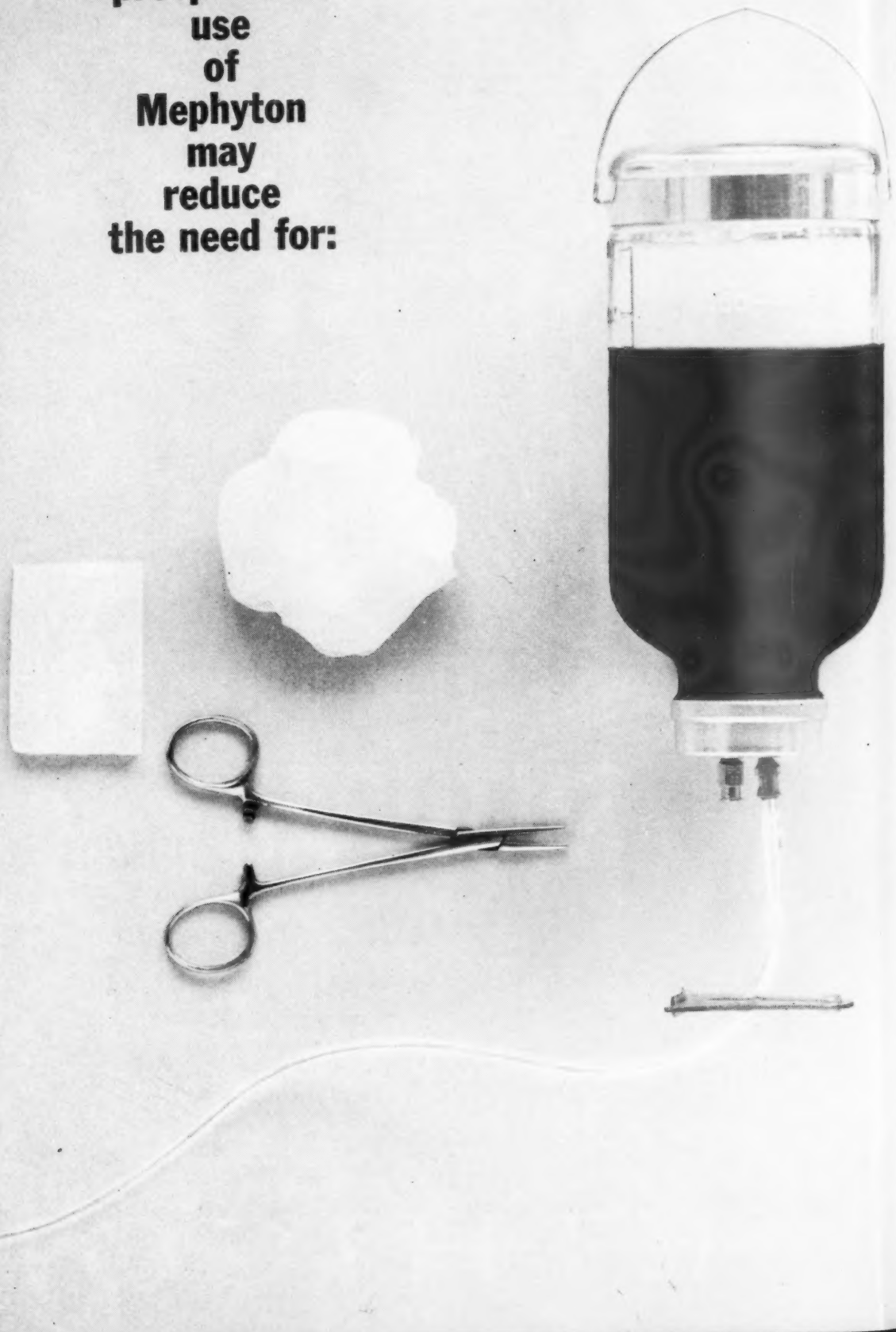

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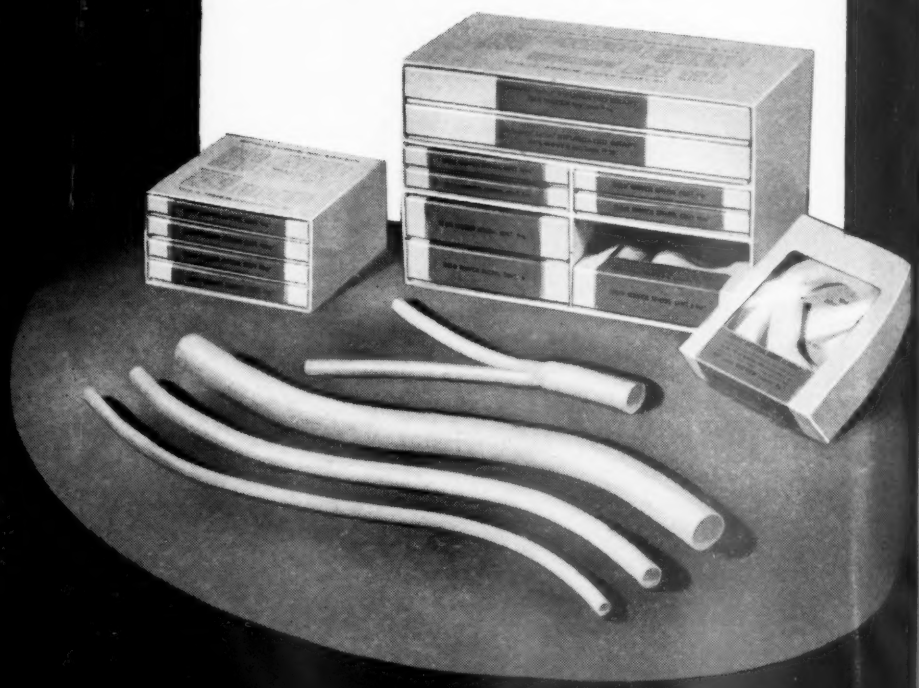

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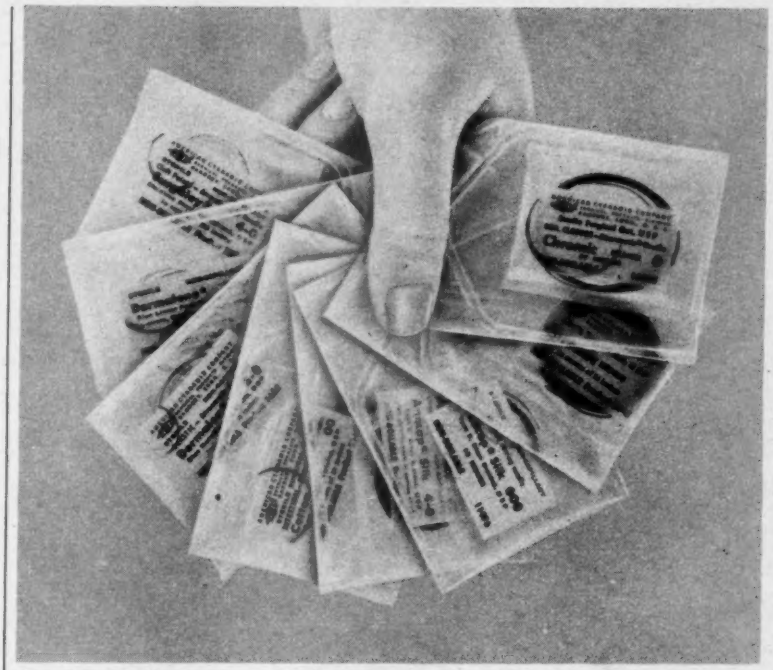

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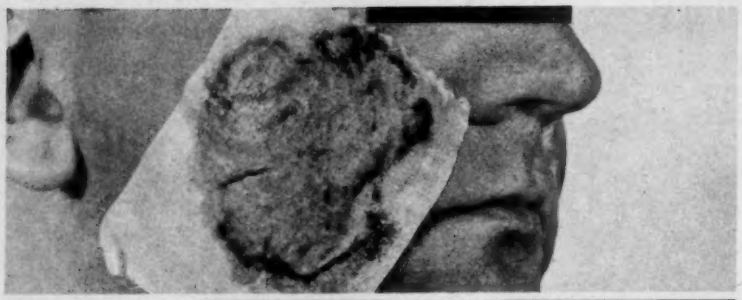

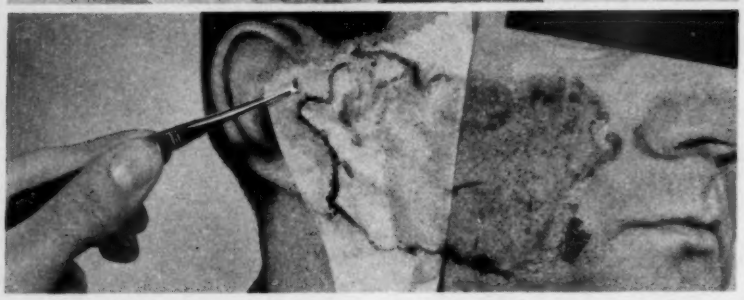

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1. Thomson, J. E. M.: Am. J. Surgery 91:413, 1956.

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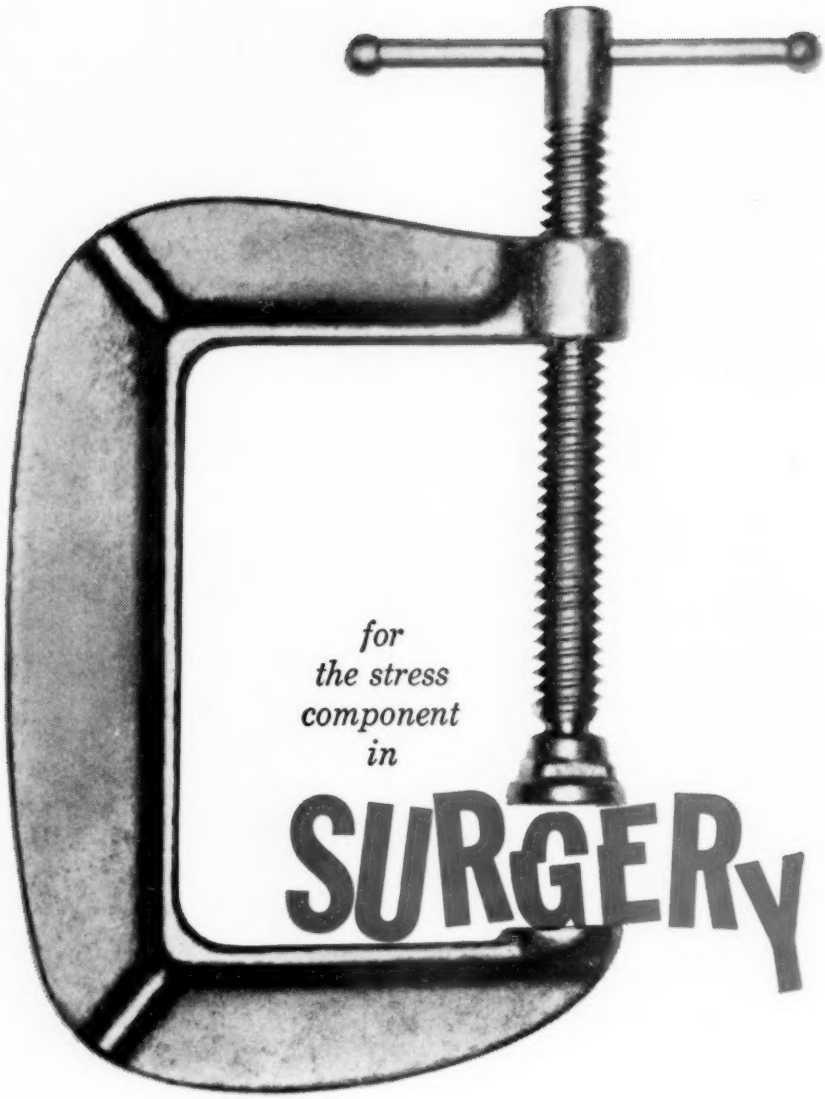

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1. Kreiger, H., et al.: Surgery 44:138 (July) 1958.
2. Priestly, J. T.: Surg. Gynec. & Obstet. 107:375 (Sept.) 1958.
3. Goodhart, R. S.: M. Cl.n. North America 40:1473 (Sept.) 1956.

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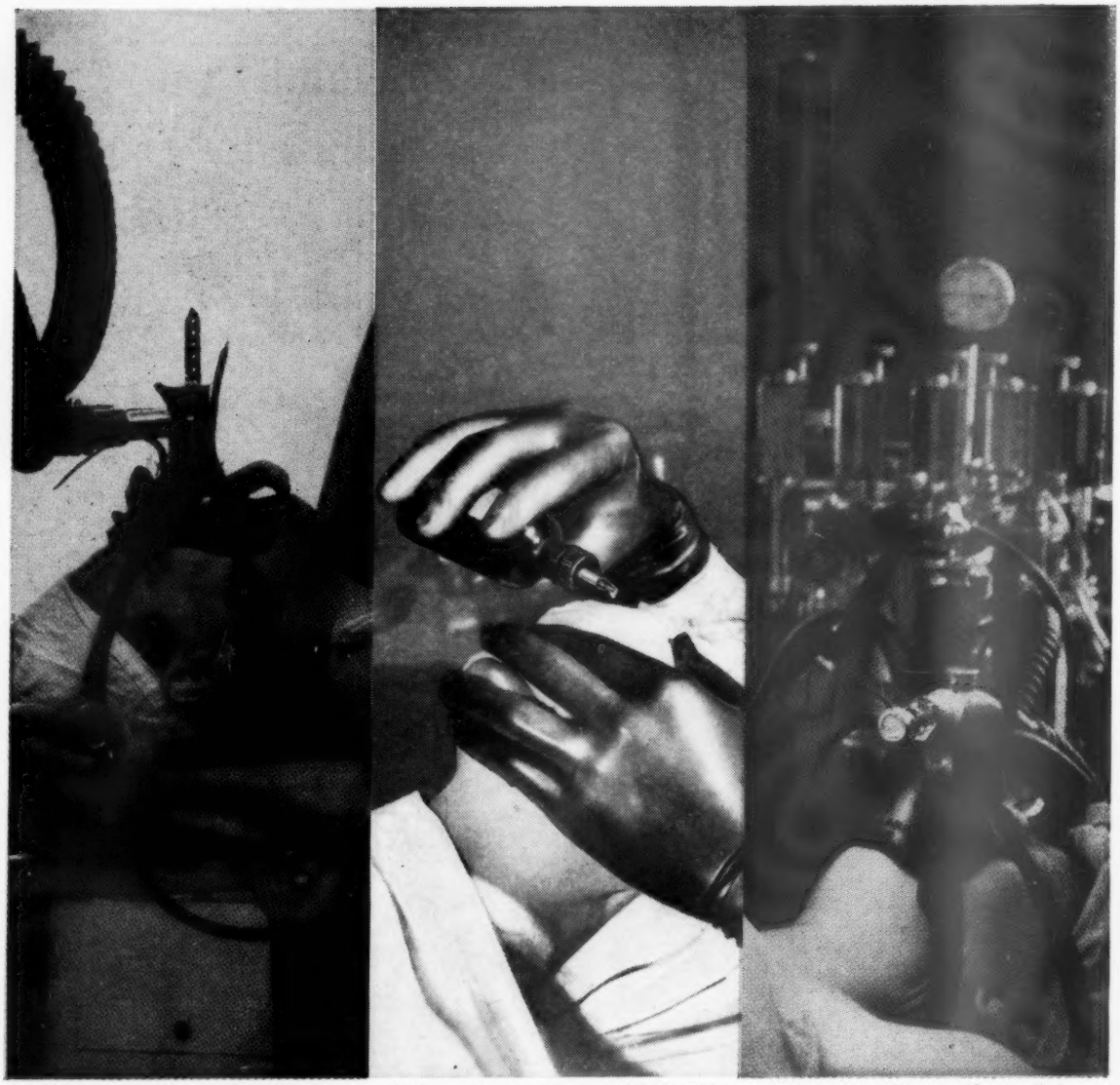

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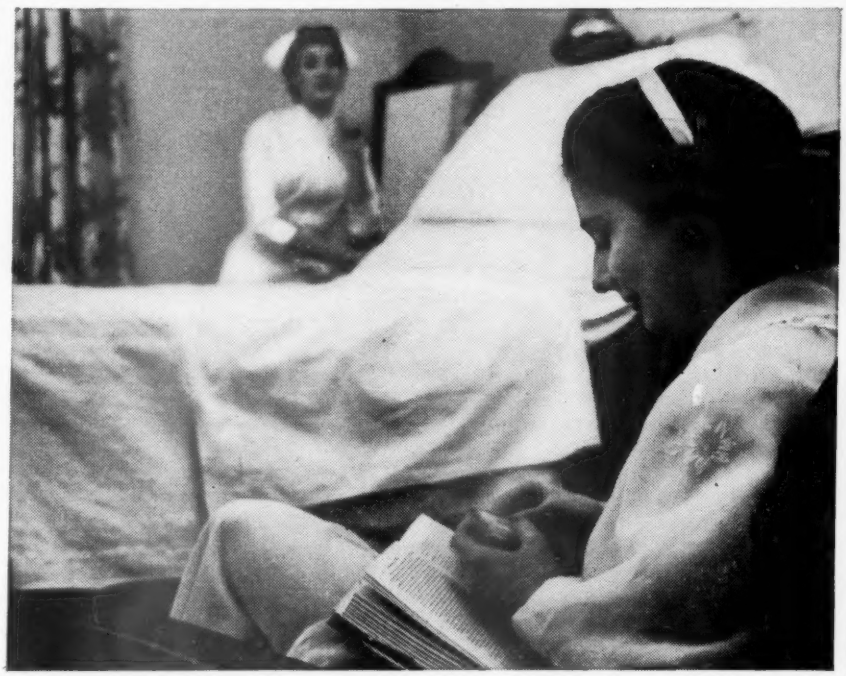

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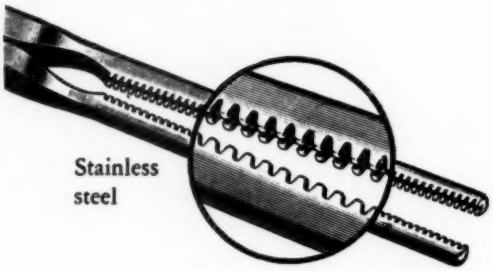

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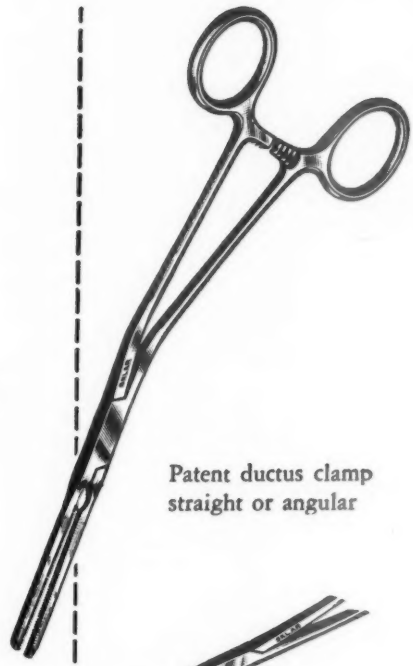

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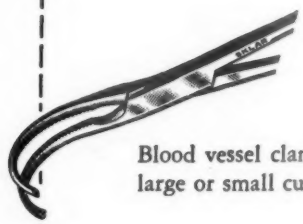
vena cava clamp



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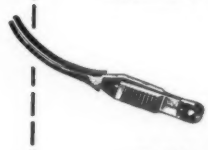

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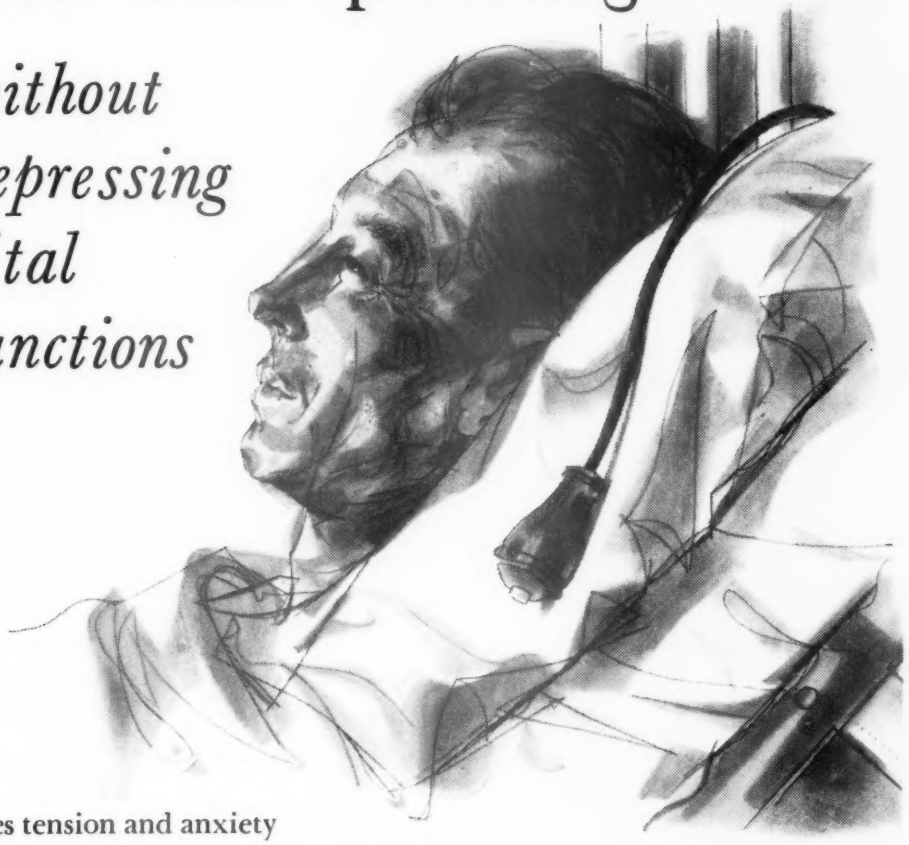

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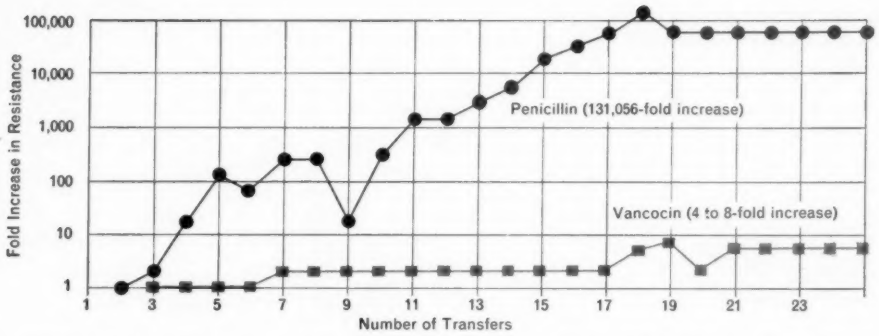

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